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THE BLOOD SUPPLY OF THE OPTIC NERVE AND ITS CLINICAL SIGNIFICANCE

THE SECOND FRANCIS I. PROCTOR LECTURE*

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The finer details of the blood supply of the optic nerve are not adequately covered in the textbooks or even in the reference books of ophthalmology. The larger vessels and the general anatomic relationships are discussed to be sure, but there is scant mention of their important terminals or loops. Since it is these latter that have ultimately to do with the nutrition of the optic-nerve tissue, they warrant more study in order to obtain the information we need in the interpretation of clinical findings. Schwalbe in 1887, Kuhnt in 1879, Leber in 1903, and Magitot in 1908, concerned themselves with the problem and by injection experiments demonstrated many factors that were confirmed by later observers. These in turn have added and refined our information so that a reasonably clear picture develops. It is the purpose of this paper to review the anatomy of the optic nerve and its blood supply and to touch on a few of the lesions with which ophthalmologists are concerned.

GROSS ANATOMY OF OPTIC NERVE AND CHIASM

It is necessary for a better understanding to consider briefly the gross anatomy of the optic nerve and chiasm. Schaeffer and deSchweinitz, in classic articles, have shown that there is a considerable variation in the anatomic relationship between the optic chiasm and the diaphragma sella, the sella turcica, the circle of Willis, the internal carotid arteries, the cavernous sinus, and the third ventricle. The length of the intracranial portion of the optic nerves have also been shown to vary considerably and, in association, the intracranial portion of the ophthalmic artery likewise shows varying lengths in different specimens. However, in spite of all of these variations in approximately 75 percent of individuals, the relationship of the optic chiasm to the adjacent structures is reasonably constant. The variations must be borne in mind for they may explain anomalous and puzzling clinical findings; for example, bizarre field defects in pituitary disease or bizarre pressure effects on the chiasm from calcified or aneurysmic lesions in the adjacent vessels of the circle of Willis.

According to Wolff, "the optic nerve, ensheathed in pia, runs as a flattened band from the antero-lateral angle of the somewhat quadrilateral chiasm forwards and outwards to the optic foramen. About half way along this course it receives a covering of arachnoid.

"Becoming more oval and acquiring a dural covering it traverses this foramen or canal and enters the orbit. As a rounded cord, it now runs forwards and slightly outwards and downwards in a somewhat sinuous manner (to allow for ocular move-

* Presented before the University of California Medical School, September 18, 1947.

ments) and is attached to the back of the eyeball, just above and at a 3-mm. internal to its posterior pole.

"It total length is 5 cm.; the intracranial portion being about 1 cm., the intracanalicular portion 6 mm., the intraorbital 3 cm., and the intraocular 0.7 mm.

RELATIONS

"*(a) Intracranial portion.* The nerve lies at first on the diaphragma sellae, which covers the pituitary body, then on the anterior portion of the cavernous sinus. Above it is the anterior perforated substance, the medial root of the olfactory tract, and the anterior cerebral artery, which crosses it from without inwards. The internal carotid artery is at first below, then lateral. The ophthalmic artery usually comes off the internal carotid under the middle of the optic nerve, but since its course here is antero-posterior, and that of the nerve outwards as well as forwards, it may appear at the inner border of the nerve before it eventually passes laterally. At any rate, in this first portion of its course it is nearer the inner border than the outer. The nearer the origin of the artery is to the optic foramen, the nearer the inner side of the nerve is placed, and vice versa.

"*(b) Canalicular portion.* The nerve is surrounded by its membranes, dura arachnoid and pia, but it should be carefully noted that above, these are firmly united to each other, to the periosteum and to the nerve. This forms a point of fixation of the nerve, which otherwise might easily be pushed backwards and forwards in and out of the cranium and thus liable to injury (Schwalbe). This fixation of the nerve also accounts for the fact that, in the optic canal, the cranial subarachnoid space only communicates with that around the optic nerve below.

"The ophthalmic artery crosses below the nerve in the dural sheath to the lateral side. It leaves the dura at or near the anterior end of the canal.

"Medially, the optic nerve is in relationship with the sphenoidal air sinus or a posterior ethmoidal air cell, from which it may be separated by a thin plate of bone only. Sometimes the sphenoidal sinus or posterior air cell may invade the roots of the lesser wing of the sphenoid, or even the wing itself. The nerve is then surrounded by air cells."

Onodi, in 1910, presented a monograph on the subject of "The Optic Nerve and the Accessory Sinuses of the Nose." In it he showed a number of beautiful and instructive specimens in which the optic nerve either lay bare in the sphenoid sinus, or in the posterior ethmoidal cells, or in very close proximity to these structures, separated therefrom, often by a very thin, bony wall. His work is perhaps forgotten for the most part, but bears careful study. The influence of sphenoiditis or inflammation of the posterior ethmoidal cells upon the optic nerve in the production of retrobulbar optic neuritis is pretty well discounted today. Undoubtedly, however, certain cases of this disease can only be thus explained.

Herzog, carrying this work further, showed that where the nerve is separated from the sinuses by a bony wall there is great variation in the thickness and substance of this wall. Some of the specimens showed a heavy thick bone with few lacunae; others were thin and cancellous with lacunae that connected directly with the mucous membrane of the sinuses on the one hand and the dura of the optic nerve on the other.

To continue with Wolff's description of the optic nerve:

"*(c) Orbital portion.* At the optic foramen the nerve is surrounded by the origin of the ocular muscles, that of the superior and internal recti being closely adherent to the sheath. It is this connection which gives rise to the pain so characteristic of retrobulbar neuritis.

"Between it and the origin of the external rectus are the two divisions of the III nerve,

the naso-ciliary, the sympathetic, the VI nerve and sometimes the ophthalmic vein or veins. Farther forwards the muscles are separated from the nerve by orbital fat.

"The naso-ciliary, the ophthalmic artery and the superior ophthalmic vein cross the nerve superiorly from without inwards. The ciliary ganglion lies to the outer side of the nerve between it and the external rectus. The long and short ciliary nerves and arteries gradually surround the nerve as it passes to the back of the eyeball."

Circulation of spinal fluid to optic nerve

The central retinal artery, which comes off the ophthalmic near the optic foramen, runs forward in or outside of the dural sheath of the nerve. The artery will be discussed later in more detail. Before doing so, it is necessary to look into the relation of the basal cisterns of the brain and investigate the circulation of the spinal fluid to the optic nerve in all of its parts.

According to Locke and Haffziger, the cisterna basalis and the cisterna magna cerebellomedullaris are to be considered as the main pathway of the cerebrospinal fluid. The smaller spaces are channels leading from the main stream. The basal cistern is made up of pars pontis, pars interpeduncularis, and pars chiasmatis. The pars chiasmatis is the portion of the cisterna basalis located farthest forward. It extends forward a short distance and posteriorly to fill in the space between the diverging limbs of the chiasm. The pars chiasmatis also extends about the chiasm, and infundibulum, as well as continuing along the optic nerves toward the globes. It may partially envelop the pituitary body. It is the deepest portion of the cisterna basalis, an important point relating to gravity and its bearing on inflammatory stasis, as well as from a therapeutic point of view.

The superior surface of the sphenoidal sinus is marked by a groove in which lies the chiasm and origin of the optic nerves. Often very thin, it corresponds topographically with the cisterna chiasmatis. Still thinner is the bony lamella making up the roof of the posterior ethmoidal cells. It is perforated by multiple openings, assuring numerous communications between the nasal fossae and the anterior meningeal spaces.

Cerebrospinal fluid is contained not in the subarachnoid space probably, but between two layers of the arachnoid, one of which is adherent to the dura and the other to the pia. It is the various enlargements of the space between the two layers of the arachnoid that form the different cisterns. Adhesions between these two layers are responsible for the collection and stasis of cerebrospinal fluid and lead the formation of subarachnoid cysts and inflammatory bands of tissue (Vail). This condition is known as adhesive arachnoiditis and occurs any place in the central nervous system.

Weed and others have shown that cerebrospinal fluid penetrates into the most minute structure of the spinal cord and brain along the perivascular spaces. So that wherever vessels go, they are surrounded by collarettes of spinal fluid. This probably applies to the chiasm and optic nerve and one can readily see that cerebrospinal fluid may accompany the septal network of capillaries in the optic nerve particularly.

However, Behr believes that there is a separation between the intervaginal space and the perivascular connective tissue of the central vessels where they enter the optic nerve and that this separation is a complete one. Thus the lacunar system of the axial-nerve part is separated from the intravaginal space. There is no possibility that cerebrospinal fluid could enter the axial-nerve part or the perivascular spaces of the intraneural vessels. Thus free fluid in the nerve tissue itself moves absolutely separately from the free fluid in the septal tissue. Hemorrhages and edema are able to spread along the septa in the nerve stem, but they do not enter the nerve parenchyma as

long as physiologic borders (pial) remain intact.

Mortensen and Sullivan have shown by means of ingenious experiments that spinal fluid passes both into the venous channels and into the cervical lymph nodes, as originally demonstrated by Key and Retzius, in 1876. Thorium dioxide, injected into the subarachnoid space of animals, passes rather readily into both the superficial and deep lymph nodes, which are visible by X ray. Batson confirmed the work of Mortensen and Sullivan, tracing the passage of the opaque mass from the subarachnoid space along the olfactory nerves to the nasal mucosa and then by way of lymph vessels along the pharynx to the upper deep cervical nodes. LeGros Clark (cited by Batson) has demonstrated that a flow in the reverse direction is possible. It has long been known that a free vascular connection exists between the nasal mucosa and the intracranial contents.

Therefore, it appears entirely probable that the protection to nerve structure by bone is only of structural significance, and bone presents no barrier to hematogenous or lymphatogenous infections, toxins, or influences.

Magitot (1908), by injection experiments, was led to believe that the intracranial and intracanalicular portions of the optic nerve have a separate blood supply from that of the intraorbital portion. The orbital portion of the nerve is supplied by branches of the ophthalmic artery, while the intracranial and intracanalicular portions are supplied by meningeal twigs arising from branches of the internal carotid, anterior cerebral, and anterior communicating arteries. Thus while the central retinal artery, which is derived from the ophthalmic, sends small nutrient vessels from the center of the nerve to the periphery, on the other hand the nutrient vessels of the canalicular and intracranial portions of the nerve are from the periphery to the center. These nutrient twigs are end-arteries. This explains the ease, if Magitot is correct in his premise, with which the papillomacular bundle, very delicate and highly differentiated, can be affected by endogenous toxins or infections. The basal cisterns thus become a common meeting ground between nerve parenchyma and blood, on the one hand, and the nasal sinuses, lymphatic systems of the sinuses, and the skull, on the other.

Vascular supply of the optic nerve

Arterial

Our understanding of the vascular nutrition of the optic nerve, although still somewhat confused, has been greatly clarified by the experimental work of Beauvieux and Ristitch (1924) and particularly of Eugene Wolff (1939) and Behr (1935). These authors have shown some discrepancies in their findings, but not enough to interfere with a reasonable interpretation.

The intracranial portion of the optic nerve is supplied chiefly by minute branches, inferiorly, from the internal carotid, sometimes the ophthalmic; superiorly, from the anterior cerebral and anterior communicating arteries. That there can exist much variation in individual cases, is evident from the work of Schaeffer and deSchweinitz. These minute vessels penetrate the pia, pass into the nerve tissue, and form the septal network of the nerve. In doing so the pial sheath is invaginated and forms the connective-tissue covering of the septal capillaries. The fibrous tubes making up the septa are not absolutely closed, according to Behr, but show spaces or small openings communicating with other bundles. The septa are separated from the nerve bundles by glial fibers. Metabolism is regulated principally or exclusively by the bordering glial membranes and the fibers connecting them. Nutriment and metabolites coming from the vessels pass first the perivascular cerebrospinal space, then through the connective-tissue covering. Fluid is collected in this mesodermal connective tissue, and what is

needed from this fluid for the nutrition of the ectodermal nervous tissue passes through the bordering glial membrane. Behr makes the point that, by this collection of free-tissue lymph fluid, a uniform uninterrupted nutrition of the nerve is maintained without quantitative imbalance.

The septa (blood vessels) of the intracranial part can be clearly differentiated between the peripheral and axial portions. In the latter, in which the papillomacular bundle is situated, the septa are thinner and more scarce (the "end-arteries" of Magitot) than in other places, and the individual optic-nerve bundles thus are larger. The nutrition of the papillomacular bundle is therefore less abundant in the intracranial part of the optic nerve than elsewhere. This anatomic finding is probably sufficient to explain the vulnerability of the papillomacular bundle.

The intracanalicular portion of the optic nerve, approximately 6 mm. long, is supplied by pial vessels arising from the internal carotid, and anastomoses with septal vessels from the orbital and intracranial portions of the nerve. Here the septal network is relatively poor. Behr also points out that in the optic canal, there are strong bridges between the optic nerve and the bone, formed by radial connections between pial sheath and dura-periosteum. These bridges are dangerous in skull fractures, injuring the nerve more often than direct injury by bone splinters.

The orbital portion is supplied by two groups of vessels derived from the ophthalmic artery. (a) Those that pierce the dura behind the entrance of the central vessels, and (b) those that anteriorly join the pial network at the site of the entry of the central vessels. Occasionally, there is found a recurrent branch of the central vessel (Wolff, Igersheimer, Abbie) which may extend backward as far as the optic foramen. Apparently, this is a rare finding.

The upper and lateral portions of the periphery of the nerve is supplied by minute branches from the ophthalmic artery, which freely anastomose with each other; with the posterior ciliary arteries at the circle of Zinn; and with the recurrent or collateral arteries that arise from the central artery as it enters the dural sheath. These latter supply chiefly the inferior periphery of the nerve.

As they pierce the dura from different directions, they traverse the subarachnoid space either at right angles or obliquely and, shrouded in meningeal tissue, form the septa.

The axial or central part of the nerve is supplied by branches from the central collateral artery (Wolff) and occasionally, as mentioned before, by a posterior axial branch from the central artery, fine branches of which anastomose with the pial network of vessels.

According to Wolff "the central artery of the retina, about 0.28 mm. in diameter, comes off the ophthalmic artery close to the optic foramen, usually in company with one or other branches, most commonly the medial ciliary artery. It runs a wavy course forward to some 10 to 15 mm. behind the globe. From this position on the under aspect of the nerve until it passes into the retina, the artery makes five bends. The first bend is found where the artery turns upwards at the right and angles, or nearly so, to pierce the dura and arachnoid, from both of which membranes it receives a covering. Having reached the subarachnoid space clothed by the above membranes, it bends forwards and also to one or the other side. After a very short course, for the artery is always cut here transversely in sections, it again bends upwards at nearly a right angle, passes through or rather invaginates the pia to reach the center of the nerve. The entering vessel is clothed by the whole thickness of the pia, and at first some arachnoid trabeculae as well, and thus takes with it the contained (pial) vessels. The fourth bend is formed where the artery passes forward in the center of the nerve. The fifth bend

is made by the terminal branches as they leave the parent vessel at the nerve head to pass into the retina.

"At about the point where the central artery pierces the dura it gives off one or more branches, which diminishes its diameter by about one third. Some of these immediately enter the pia, dividing into branches which go forwards, backwards, and circularly, and joining the pial network, send branches into the nerve. Others pass into the nerve with the central artery, running parallel with it. Although extremely well described by Kuhnt in 1879, these vessels have often been forgotten in subsequent works, and this has led to a great deal of confusion." (To emphasize the fact that they do exist Wolff suggests the name of central collateral arteries.)

"One of these vessels, larger than the rest, can be traced to the lamina cribrosa. These collateral vessels send branches into the nerve and hence get narrower as they pass anteriorly. At the point where the central artery bends forwards at the center of the nerve a branch of the collateral vessels, not of the central artery itself (Magitot) passes backwards, towards the optic foramen.

"While the collateral arteries get finer as they are traced forwards, the central artery remains the same size from its point of penetration to its bifurcation. This is due to the fact that in this portion of its course the central artery gives off no branches, or at least none of any consequence, hence in its intraneural course, takes no part in the nutrition of the nerve (Kuhnt, Behr)."

Origin of central artery

Beauvieux and Ristitch found eight variations in the origin of the central artery.

1. It springs from a trunk common to the long internal ciliary artery on its superior surface (1 in 20).

2. It arises from a trunk common to the external ciliary artery, turns around the external edge of the nerve to penetrate its superior surface (2 in 20).

3. Same as above, except the vessel penetrates the inferior surface (3 in 20).

4. It arises from a trunk that is common to external and internal long ciliary arteries and reaches the inferior surface of the nerve (1 in 20).

5. It arises from a trunk common to the long internal ciliary artery on the external or lateral edge of the nerve. It then turns around the latter and disappears in its inferior surface (5 in 20).

6. It arises from a trunk common to the short posterior ciliary artery, turns around the nerve and enters into its inferior surface (1 in 20).

7. It arises directly from the ophthalmic, external margin, after the lacrimal artery and penetrates into the nerve, sometimes in its superior surface (1 in 20) and, sometimes in its inferior surface (5 in 20).

8. It appears as the first branch of the ophthalmic and enters the nerve in its inferior surface (1 in 20).

These authors are at variance with Wolff in the conclusion that no communication exists between the central arteries and the vessels of the pia of other origin, which contribute to the vascularization of the orbital portion of the nerve, nor do they believe that there is any communication of the central artery branches and those coming from the anterior cerebrals.

The injections by Beauvieux and Ristitch of the central artery alone, have shown that the region of the lamina cribrosa is supplied by twigs from the arterial circle of Zinn (short posterior ciliary arteries). The circle of Zinn together with twigs from the central artery, supply the papilla of the optic nerve. Branches are also sent to the retina, even the fully developed cilioretinal artery. There are apparently no anastomoses between the hyaloid and sclerochoroidal systems.

Central retinal vein

The central retinal vein has numerous anastomoses with the capillaries near the papilla, the lamina cribrosa, and the choroidal system. Beauvieux and Ristitch showed that coloring matter injected into the central artery can be seen to fill the peripapillary choroidal veins, then into the vortex veins, and also into the scleral veins which contribute to form the short posterior ciliary veins. The central vein leaves the optic nerve either obliquely or parallel and then at right angles, usually proximal to the entry of the artery. From thence it becomes the venous network in the orbital fat which empties in the cavernous sinus, the superior ophthalmic vein, the inferior ophthalmic vein, or several combinations. The central vein contributes also to the plexus surrounding the optic nerve and ophthalmic artery.

According to Cone and MacMillan, the posterior central vein of the optic nerve (Kuhnt) is found in the center of the optic nerve during its course through the optic canal. It arises by confluence from numerous venules which collect blood from the orbital portion of the nerve, and it is accompanied by a strand of connective tissue which is the continuation of the pia. It leaves the stem on its lower aspect where the ophthalmic artery is found in the dural sheath. The vein then proceeds around the external margin of the nerve and empties into the cavernous sinus (Greef 1900). The finding is constant and its lumen is nearly the same size as the anterior central vein. Greef says that, according to Berlin, injury of the vessel is responsible for the hemorrhage present at times in fracture of the optic canal.

The posterior central vein of Kuhnt seems to have been neglected and almost forgotten. It may play an important role not only in injury to the nerve in the optic foramen but, by sudden compression in spontaneous subarachnoid hemorrhage, it may lead to profound and remote results in the retina.

Elschnig (1893) was the first to show that a rich anastomoses existed between the arteries arising from the external carotid and the ophthalmic artery. He conducted experiments on a cadaver and showed that after the occlusion of the internal carotid artery intracranially, water and water soluble dyes injected into the common carotid artery would, by retrograde flow appear in the distal segment of the ophthalmic artery. Walsh and King verified this important observation in a series of ingenious experiments and conclusively showed that a large collateral circulation exists between the external carotid artery and the ophthalmic artery of the same side and that there is sufficient cross circulation so that the ophthalmic artery of the opposite side may be filled from the external carotid artery.

Clinical significance

A good understanding of the nutrition of the optic nerve helps us to interpret the clinical manifestations encountered when the nutrition is interfered with. There are, of course, many gaps in our knowledge and many errors of interpretation.

The demyelinating diseases, of which multiple sclerosis is the most common, are poorly understood. There is some glimmer of hope in the recent studies of Putnam and his co-workers. These investigators bring strong evidence to bear upon the premise that thrombosis of venules are essential links in the chain of causation of multiple sclerosis and encephalomyelitides. The premise is based upon the following evidence: (1) Experimental. (2) Human cases—similar lesions in human material following thrombosis of veins of certain size or compression of a pial vein by a tumor. (3) Thrombi, usually in venules and veins, have been observed in a large proportion of cases of acute encephalomyelitis and

in the more acute lesions of multiple sclerosis by various authors over the past half century. (4) Exogenous factors—infection, pregnancy, trauma, and chilling increases the coagulability of the blood to produce thrombophlebitis. These factors correspond closely with those that appear to precipitate the onset or exacerbations of multiple sclerosis. (5) The clothing mechanism of the blood is abnormally labile in cases of multiple sclerosis. Obstructive changes (Rucker) have been observed in the retinal vessels, some of which might be interpreted as thrombotic. Abnormalities can be observed in the capillaries of the nail bed in a majority of cases.

Sheinker studied 20 cases of multiple sclerosis. In all cases the early stage of plaque formation showed positive correlation between the lesion and vascular changes that are probably thrombotic.

There is also some evidence that a tendency to thrombosis may in some measure be allergic and Foster-Kennedy has described cases of acute retrobulbar optic neuritis which were definitely allergic. Putnam and his co-workers treated 43 patients, who had multiple sclerosis, with dicouramin. The results are promising. The dramatic response to blood letting or draining of the sinuses, especially the sphenoid, that is seen in some cases of retrobulbar optic neuritis suggests that the circulation in the optic nerve has a lot to do with the condition. It is also logical to assume from the description of this circulation that has been given, that the intracranial portion of the optic nerve is the primary locus of the disease. Rønne has described a number of cases of acute retrobulbar optic neuritis where the lesion was located in the chiasm; and careful field studies by other observers including myself have supported his findings.

Similar arguments can be directed toward the pathogenesis of other forms of neuritis and the toxic amblyopias. That is to say, a suppression of function of the papillomacular bundle, more commonly in the intracranial portion, due to interference with its blood supply.

The papilledema that is seen on rare occasions in cases where only a focal infection can be found is presumably on a thrombotic basis. The removal of the focus and the establishment of collateral circulation results in improvement (Frost).

Verhoeff and Simpson report a remarkable case of tubercle in the central vein associated with periphlebitis retinalis in the other eye. Other tubercles were found in the vaginal space, in the arachnoid, pia, and in the episcleral tissue, 1.8 mm. outside of the subvaginal space. Their patient had a hemorrhagic glaucoma, the cause of which was not determined until histologic examination. The Mantoux test was negative.

Goldstein and Wexler described a case of bilateral atrophy of the optic nerve due to periarteritis nodosa. Histologic examination showed an affected postciliary artery entering the dura of the optic nerve and proceeding into the region of the lamina and anterior segment of the nerve. The optic disc contained many inflammatory round cells and glial tissue. The infiltration extended behind the lamina and the nerve fibers were atrophic. In Von Herrenschwand's case the periarterial nodes occurred particularly when the ciliary artery altered its direction, such as at its entrance into the sclera, its course through the lamina cribrosa, and on its course from the sclera toward the ciliary body.

The optic atrophy of tabes undoubtedly is associated with a nutritional disturbance of the nerve fibers due to the syphilitic involvement of the blood vessels, as Moore and Woods have so ably argued. Its relationship to syphilitic arachnoiditis has not yet, however, been entirely disapproved. It is entirely possible that the pial network of vessels of the optic nerve, especially in its intracranial portion, may be the first site of the lesion, thus explaining the peripheral

contraction of fields of vision in tabes. Stargardt found, in 10 cases of syphilitic optic atrophy, earliest change in the chiasm and intracranial portion of the optic nerve. There was evidence of inflammation in the pial investment and some gliosis of the superficial parts of the nerves. He noted that the myelin sheaths became degenerated before the axis cylinders. Behr, in discussing the careful study of nine cases of optic atrophy due to syphilis, noted that the primary change was an inflammatory process in the connective-tissue system of the intracranial portion of the nerve and of the blood vessels in relation to this. The myelin sheaths were affected before the axis cylinders. Greenfield and Epstein corroborated Stargardt's observations fully and remarked that the superficial fibers in the optic nerve were first affected, that the myelin was more affected than the nerve fibers, and finally that inflammatory changes in the meninges were much more evident in the intracranial than in the intraorbital portion of the optic nerves.

Gradle (1917), in discussing glaucomatous cupping and atrophy of the optic nerve in cases where the intraocular pressure was never elevated, regarded it as a special disease that began as an optic neuritis with formation of the cavernous changes in the optic nerve described by Schnabel. Loewenstein described vascular changes (thrombosis) in the optic nerve which led to cavernous degeneration, calcified drusen, and sclerotic plaques. He and Garrow also described further examples of this condition which they termed spongy degeneration and cavitation of the optic nerve. Knapp, Elwyn, and others believed that their cases showing the clinical picture of glaucoma without ocular hypertension were secondary to compression by calcified carotid arteries upon the optic nerve. Recently Rintelen, in a classic article on the arteriosclerotic atrophy of the optic nerves, described the histologic findings in 6 out of 35 cases. The atrophic conditions in the nerve were localized chiefly in the anterior third of the nerve, in the part that contains the central vessels. He also found in one case an area that he called "red malacia." Arteriosclerosis of the central artery has little effect on the nerve. The damage is due to the sclerosis of the small nutrient vessels. Subsequent to this local malnutrition, cicatriced areas occur with disappearance of the nerve fibers and secondary glial proliferation. He believed that the arteriosclerotic malacia of the optic nerve is of much more importance than the calcification of the carotid arteries. He distinguishes three forms of arteriosclerotic atrophy of the optic nerve: (1) A benign, slowly progressive, diffuse atrophy, especially limited to the periphery of the nerve. This occurs in the aged and is clinically marked by a moderately cupped and pale optic disc. (2) A malignant, rapidly progressing atrophy which often shows exacerbations, occurring mostly in young patients with arteriosclerosis or hypertension. This form is caused by the malacious areas in the anterior part of the nerve and may have the clinical aspect of a glaucoma without ocular hypertension. (3) A clinically less important atrophy of the optic nerve caused by a direct compression of the nerve by calcified carotid arteries.

It is entirely probable that vascular disease of the nerve as previously described may account for the progress of cupping, atrophy, and field changes that frequently continue after the ocular hypertension has been quite controlled in true glaucoma, especially in the aged.

Drusen bodies in the papilla are hyalin formations. They are formed locally and not directly from surrounding tissue. The first anatomic signs of drusen formations are found in the smallest vessels in front of the lamina cribrosa. Before hyalin is present, the walls of these vessels become thickened. Drusen bodies are found else-

where in the optic nerve and may, by pressure or interference with the nutrient vessels, disturb the function of the nerve fibers. Tuberose sclerosis is closely allied to this process.

The retinal and preretinal hemorrhages found in cases of subarachnoid hemorrhage have created much speculation as to their origin and mode of production. The evidence is pretty conclusive that there is no direct extension of blood through the optic disc from the distended hemorrhagic vaginal sheath. Ballantyne, whose recent studies have thrown further light upon this condition, believes that, while it may be a physical possibility for blood to pass through the optic foramen, it does so only in exceptional cases. He as well as Bucklers and Greear and others think that the sudden rise of intracranial pressure produces a venous stasis which in turn leads to hemorrhages throughout the orbital and ocular structures. Hemorrhage occurs in the subdural and subarachnoid spaces, and the blood in the latter situation, by putting pressure and tension on the central retinal vein, accounts for the retinal and vitreous hemorrhages.

It must be considered also that the pressure upon the posterior part of the globe by the distended subarachnoid space compresses the veins that so freely anastomose around the optic nerve at its insertion into the globe. The role of the posterior central vein of the optic nerve in the production of these retinal hemorrhages has not been studied, so far as is known.

It has been pointed out that there is a rich anastomoses between the arteries arising from the external carotid and the ophthalmic arteries. This explains the number of cases of retained vision where the ophthalmic artery intracranially has been obliterated surgically in the treatment of vascular aneurysms of the circle of Willis (Dandy, Adson).

The cases of blindness and optic atrophy occurring rather infrequently in head injuries require some comment. There is no question but that there is complete degeneration of the axis cylinders both above and below the lesion in severe cases. Most all authors agree that the primary site of injury to the optic nerve lies in the intracanalicular portion and sometimes in the intracranial portion of the nerve, owing to their immobility, and this position of the lesion corresponds with the appearance of pallor of the disc from 14 to 21 days after the injury. The damage to the nerve may be the result of (a) subvaginal hemorrhage within the optic foramen, with pressure on or tearing of the nutrient vessels of the optic nerve, (b) intraneural hemorrhage, (c) fracture, with perhaps tearing of the nervous tissue, (4) later by callus formation within the optic foramen, following a fracture through the anterior clinoid (Turner, Lillie and Adson, Rodger). The role of the posterior central vein in this condition has been mentioned (Berlin), and it must take an active part in the associated hemorrhages from the nutrient and septal hemorrhages that undoubtedly occur at the time of the injury.

These clinical conditions are but a few of the important lesions affecting the optic nerve that a knowledge of the minute details of the nutrition of the optic nerve helps to interpret. Further studies are necessary and it is hoped that this paper will serve as a stimulus to those studies, particularly experimental ones.

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A STUDY OF THE ANGLE OF BIFURCATION OF RETINAL VESSELS*

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It is often stated that in sclerosis of the retinal vessels the arterial branches diverge from the stem vessel at an acute angle. Lurje[1] mentions that in sclerotic states in which hypertension is present the arteries are usually straight and branch acutely.

Friedenwald[2] (using the evidence presented by Hertel and by Coats that the neural portion of the central artery shows more sclerotic changes than the retinal portions) states that the straightness of the retinal arteries and their tendency to branch at acute angles must be due to longitudinal shrinkage in the central vessel behind the disc, which draws the branches of the first and second order onto the disc.

To add credence to this hypothesis, he examined the fundi of two groups of elderly arteriosclerotic patients—one group in whom the retinal arteries were straight and narrow and one group in whom they were straight and tortuous—counting the number of major branches which crossed the disc margin. The group in whom the retinal arteries were straight and narrow showed an average of 5 to 6 major branches crossing the disc margin while the group in whom the retinal arteries were straight and tortuous showed an average of slightly better than four. This appears to be the only evidence in the literature to substantiate this oft-quoted statement.

Friedenwald, however, believes that, in the condition which he designates arteriolar sclerosis, the angle of bifurcation is more obtuse. In his Doyne Memorial Lecture he stated: "The larger retinal arteries in early cases (of arteriolar sclerosis) are full and tortuous and often show a widening of their reflex band associated with a coppery appearance of blood column. . . . In late stages of this condition the larger arteries also show marked lesions, such as beading, increased visibility of their walls, and even diffuse constriction of caliber. A point of distinction between these cases in their late stages and those of the group next to be described (arteriosclerosis with hypertension) is that the larger arteries in arteriolar sclerosis usually remain tortuous and branch at right angles even when they have become greatly narrowed, while in the next group they are generally quite straight and branch at acute angles." However, no evidence is presented to support this concept.

PLAN OF STUDY

In order to assemble some specific data on this subject, a plan of study was drawn up. The angles were measured from photographs of normal and arteriosclerotic fundi

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for comparison. Bedell's *Atlas of Fundus Photographs*,[5] was used since it was thought that the results would be less prejudiced than if studies on a selected series of pictures were made. Those photographs were selected in which the branches of the retinal artery were most clearly in focus. Because of the lack of detail in these reproductions, it was found impossible to measure accurately the angle by direct application of a protractor. Therefore, each photograph was enlarged by means of a reflection projector on a screen of white paper. The arterial pattern was traced on this paper. In addition to enlarging the photographs from 3.5 cm. to 23 cm., this procedure proved advantageous in that it transferred the photograph to white paper where trigonometric calculations could more readily be made.

Calculation of Angles

In calculating the angles at the first, second, and third bifurcations of the central arteries, straight lines were drawn from the junction of the branches and stem, following the centers of the vessels. In this way tortuosity of the two arterial limbs distal to the bifurcation did little to influence the fairly accurate construction of the angle. Each line was extended to exactly 5 cm.; the ends were joined by another line, creating an isosceles triangle. A perpendicular was dropped from the vertex of the angle to this line which necessarily bisected the angle.

Using elementary trigonometry, either half of the bisected angle was readily calculated; that figure, multiplied by two, revealed the size of the angle in question. In this way, 70 angles of normal fundi and 23 angles of arteriosclerotic fundi were measured. Contrary to expectations, the averages showed a normal average angle of 70 degrees and an arteriosclerotic average angle of 82 degrees.*

In other words, in this small series, and by the methods employed, the arteriosclerotic vessels showed a more obtuse angle than did the normal vessel angles. This result was quite contrary to the generally accepted statements found in the literature already referred to with the possible exception of that made by Friedenwald. Individual variation in the size of the angles in different fundi was found to be very great in both normal and arteriosclerotic fundi, ranging from a low of 37 degrees to a high of 106 degrees in normal fundi, and from a low of 45 degrees to a high of 108 degrees in arteriosclerotic fundi.

Comments

Interesting is the fact that in those few cases where several bifurcations were measured from the same fundus, individual variations were not nearly so marked. Of 18 normal fundi in which several angles were measured in each, in 9 the angles differed by less than 10 degrees and in 15 by less than 20 degrees; of the 6 arteriosclerotic fundi so measured, in 2 the angles differed by less than 10 degrees and in 5 by less than 20 degrees.

Although every precaution was taken to standardize the technique there were many instances in which the construction lines following the long axis through the center of the vessels offered a number of choices; thus, others repeating the study might arrive at somewhat different averages. However, since this error occurs equally in measuring both normal and arteriosclerotic fundi, it seems reasonable to assume that others might find a similar ratio between the two

* The standard deviation of the measurements for the bifurcation angles of 70 normal retinal arteries is 14.2; for the 23 angles of bifurcation of sclerotic arteries the standard deviation is 14.5. The standard errors are, therefore, 1.7 and 3.0, respectively. For normal arteries the angle of bifurcation may be given as 69.8 degrees ± 1.7 degrees; for the sclerotic arteries, 81.5 degrees ± 3.0 degrees. The standard error of the difference is 3.5, and the ratio of the difference of the means to the standard error of the difference is 3.3, indicating a significant difference between the two groups.

groups of fundi. In several cases, the outline of the retinal vessels was difficult to visualize; perhaps in this way error might have been introduced.

Discussion

As mentioned earlier in this paper, Friedenwald stated that longitudinal shrinkage of the central artery behind the disc was responsible for the acute angles reputedly found in retinal arteriosclerosis. It seems equally convenient to explain the more obtuse angles found in this series by the converse condition; namely, a longitudinal stretching of the central artery.

Thoma[3] believed that the first change in arteriosclerosis was a loss of the normal elasticity and contractability in the walls of the arteries; consequently the arteries become lengthened. Since no increase occurs in the size of the eye, the elongated arteries must adapt themselves to the environment by becoming tortuous.

Duke-Elder[4] also believes that the elongation of the arteries causes an increased degree of tortuosity but ascribes the elongation to the hypertrophic thickening of the vessel walls. He states that if the thickness of the walls is increased, the length of the vessel must also increase, for it appears unlikely that the additional tissue would be deposited in the vessel wall in one plane only.

Perhaps this arterial lengthening, which is thought to be the cause of tortuosity, is the cause of the more obtuse angles that this short series of measurements seems to indicate are present in arteriosclerosis. In order to determine whether this longitudinal stretching does occur, an attempt was made to measure the length of the central artery, on the fundus photographs, from its point of emergence from the disc to its first bifurcation. However, because most of the vessels in the disc area were out of focus and therefore poorly defined, no accurate measurements could be made.

One might more reasonably postulate a shrinkage of the retina itself with retraction toward the nervehead. This would tend to widen the angle of the relatively stiff vessel tree as the branches would be flexed away from the more resistant trunk vessel. Moreover, the branches are anchored to the retina by their capillaries; whereas, we know that there is a capillary free zone about the arterial trunks. Retraction toward the periphery would not be expected because we know that the retinal periphery in arteriosclerotic subjects shows degeneration, cyst formation, and other signs of atrophy and thinning.

It is evident that while the results are quite contrary to the generally accepted statements, they appear to be in accord with Friedenwald's observations on arteriolar sclerosis. However, since Bedell deals only with arteriosclerotic fundi in his *Atlas,* we are able only to report on this condition. Whether perhaps a few or many of Bedell's cases are in reality cases of arteriolar sclerosis as defined by Friedenwald is an open question; certainly one cannot tell from the brief descriptions or from the photographs. We are able, therefore, only to say that in this study of arteriosclerotic fundi, as selected by Bedell, it was found that the arterial branches came off more obtusely than did the arterial branches of normal fundi of a larger series.

The fact that in normal fundi the angles vary from 37 to 106 degrees, and in arteriosclerotic fundi from 45 to 108 degrees precludes the possibility of using the size of the angle of bifurcation as an aid in diagnosis. However, if serial funduscopic photographs were taken of individual cases over a period of years, perhaps then the measurement of the angle of bifurcation by the method described in this study might prove a reliable index of the progress of sclerosis.

Summary

Using Bedell's funduscopic photos the angle of bifurcation of 70 normal retinal

arteries and of 23 arteriosclerotic retinal arteries was measured. The average angle in the normal fundi was found to be 70 degrees and in the arteriosclerotic fundi 82 degrees; this appears to be quite contrary to the generally accepted statements found in the literature. A brief discussion of the possible mechanisms responsible for the more obtuse angles in arteriosclerosis is presented.

Acknowledgement is made to Dr. John N. Evans for his encouragement and guidance in the preparation of this paper.

Henry, Pacific and Amity Streets (2).

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ON THE MECHANISM OF PRODUCTION OF MASSIVE PRERETINAL HEMORRHAGE FOLLOWING RUPTURE OF A CONGENITAL MEDIAL-DEFECT INTRACRANIAL ANEURYSM*

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Two possible explanations have been offered for the preretinal hemorrhages commonly associated with ruptured congenital medial-defect aneurysms of the cerebral arteries. In one of these, the older, it is postulated that blood passes by direct extension from the subarachnoid space along the optic nerve through the lamina cribosa to infiltrate the retina[1, 2] (fig. 1). In the other, a more recent and generally accepted explanation, extravasated blood is thought to pass along the perineural or intervaginal space of the optic nerve and to constrict the central vein of the retina as it leaves the nerve,[3, 19-21] thus producing marked engorgement and eventually rupture of the preretinal veins (fig. 2). We have recently had the opportunity to make a detailed study of a case of preretinal hemorrhage of the type in question. In reviewing the anatomy of the eye especially with reference to the vascular arrangement, we have been impressed by certain venous relationships which appear to serve as an entirely satisfactory and quite probable basis for a third possible explanation of the intraocular hemorrhage (fig. 3).

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Case report

History. The patient, a 38-year-old white housewife, was in good health until the early morning of the day of death, at which time she complained of sudden, severe pain in the frontal region of the head. This was followed by vomiting and convulsions. She was hospitalized 2½ hours after onset of the pain. There was no history of trauma or of similiar episodes. Her mother was said to have died of a "stroke" two years previously.

Examination. The patient was semiconscious, uncoöperative, and responded

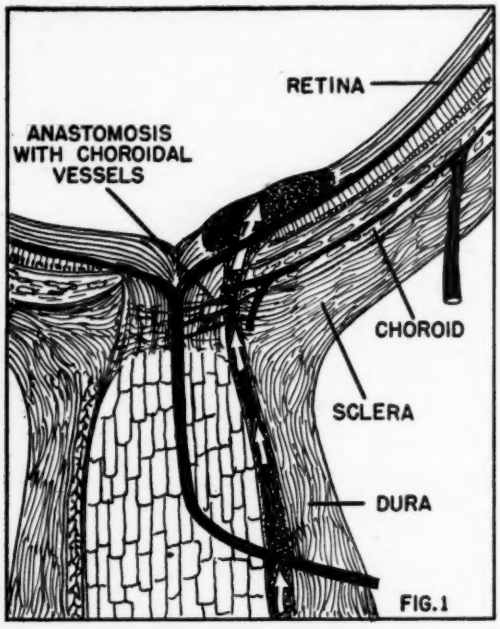

Fig. 1 (Miller and Cuttino). Diagram of mechanism of production of preretinal hemorrhage, as postulated by Symonds. According to this hypothesis, blood in the intervaginal space was thought to be of sufficient pressure to traverse the lamina cribrosa and appear in the retina.

poorly to questions. Blood pressure was 125/80 mm. Hg, pulse 60, respirations 24 per minute, and temperature 36.6°C. The skin was cold and clammy. She moved her right arm about aimlessly, but did not move her left arm. Repeated extensor spasms followed episodes of hyperventilation.

Eye Findings. A slow lateral nystagmus of the right eye was present. The right pupil was larger than the left, and both were fixed. There was a fixed left lateral gaze. Funduscopic examination showed marked protrusion of the right optic disc with a large flame-shaped hemorrhage virtually obliterating the temporal half of the right fundus. The left eye was normal. Right facial paralysis and ptosis of the right eyelid were questionable. The reflexes on the left side of the body were slightly reduced with a positive Hoffman's sign on the right.

Laboratory findings. The pertinent laboratory findings were as follows: Hgb. 8.3 gm.; W.B.C., 31,450, R.B.C., 3,480,000; platelets, normal; spinal fluid grossly bloody, contained 1,300,000 R.B.C. and 120 W.B.C. The initial cerebrospinal-fluid pressure was 570 mm. and the final pressure, 230 mm. of water.

Course of disease. Shortly after admission an extreme extensor rigidity similiar to that of the decerebrate animal followed one of the convulsions. The pupillary reflex returned, and reëxamination revealed that bilateral Hoffman and Babinski signs, ankle clonus, and equal but hyperactive reflexes had developed. Blood pressure was now 170/110 mm. Hg. The patient grew progressively weaker and became comatose. Secretions collected in the upper respiratory tract, requiring frequent aspiration. The pulse rose to 152 per minute and the temperature to 39°C. Terminally the respiration movements became slow and deep. The patient died 12 hours after onset of the severe frontal headache.

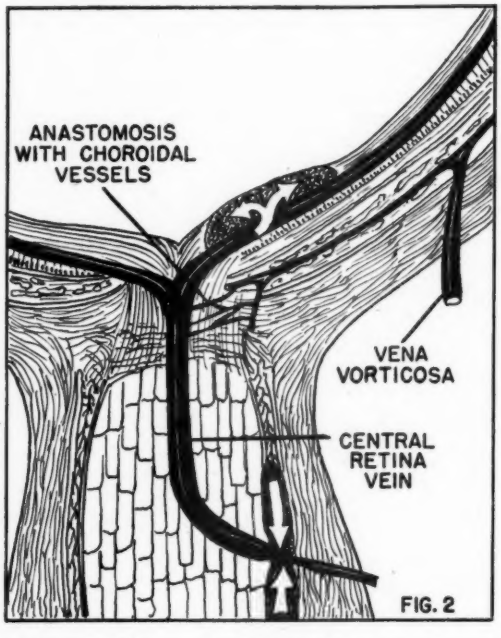

Fig. 2 (Miller and Cuttino). Diagram of mechanism of production of preretinal hemorrhage, proposed by Paton and Holmes. Here the hemorrhage results from stasis produced by compression of the central retinal vein as it leaves the optic nerve.

Autopsy findings. When the cranial cavity was opened at autopsy, the dura was under increased tension. Attached to its right under surface was a meningioma, 8 mm. in diameter. Over the convexity of the brain was a diffuse subarachnoid hemorrhage. The inferior surface of the brain likewise was covered by hemorrhage; this was especially prominent in the vicinity of the right internal carotid artery and on the inferior surface

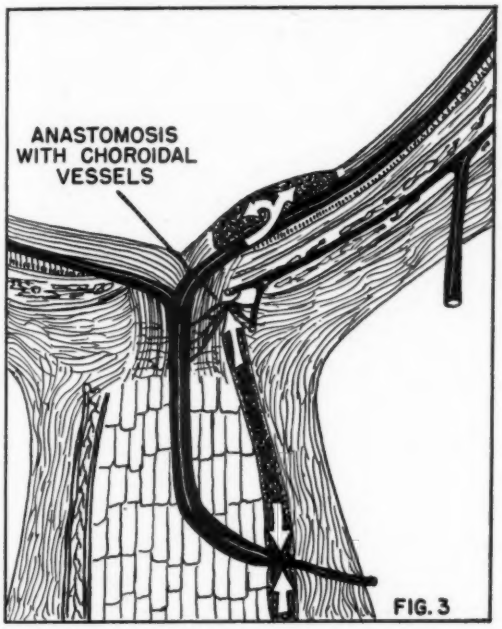

Fig. 3 (Miller and Cuttino). Diagram of the mechanism of production of preretinal hemorrhage, as outlined in Figure 2, but with the modification of compression of the choroidal anastomosis, as suggested in the text.

of the right frontal lobe. The right internal carotid artery was dilated throughout its intracranial course. At the common site of origin of the anterior cerebral, middle cerebral, and posterior communicating arteries was a saccular aneurysm, 1 cm. in diameter, with an anterolaterally situated rupture in its thin wall. Corneal sections of the brain revealed a 2-cm. deep penetration of the temporal lobe, directly opposite the aneurysm, by hemorrhage.

Nervous-system dissection. The cranial

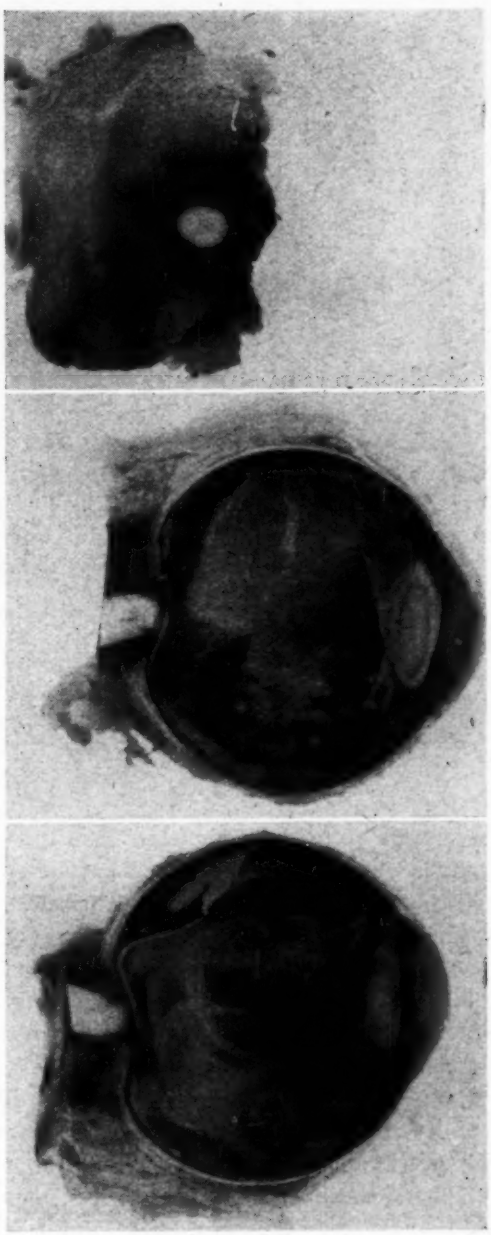

Fig. 4 (Miller and Cuttino). Right eye on section showing preretinal hemorrhage and hemorrhage about the optic nerve. The hemorrhage about the optic nerve is not continuous with the hemorrhage of the retina.

nerves were not remarkable except for the right optic nerve. This was surrounded by blood as it entered the optic foramen. There was also clearly defined hemorrhage in the central portion of the nerve. As demon-

Fig. 5 (Miller and Cuttino). Right eye showing preretinal hemorrhage confined within the retina and the relationship of the intervaginal hemorrhage to the lamina cribrosa. Hematoxylin and eosin (×17).

strated by serial cross sections at 2-cm. intervals, the latter was confined to the intracranial segment of the nerve. Throughout its orbital course the nerve was surrounded by blood, which lay in the intervaginal space. Hemisection of the right eye showed hemorrhage in the nasal half of the retina, which did not connect with that in the intervaginal space of the nerve (fig. 4). The left eye showed nothing unusual. Further dissection of the central nervous system was noncontributory.

The somatic organs showed an early pneumonia, left ovarian cyst, and right ventricular dilatation.

Microscopic findings. In microscopic preparations the aneurysm was found to be a fibrous connective-tissue sac whose thin walls contained neither muscle nor elastic tissue. Interrupted serial sections of the optic nerve and eye were prepared. The hemorrhage in the center of the optic nerve was thus shown to be small and confined to the intracranial segment of the nerve. The hemorrhage in the subarachnoid and subdural spaces was traced uninterruptedly throughout the course of the nerve to an abrupt termination at the lamina cribrosa (fig. 5). There was no connection between the hemorrhage in the intervaginal space of the optic nerve and that in the retina. The central retinal artery and vein were not constricted as they left the optic nerve, nor were they dilated in their intraneural course. The optic nerve was edematous. Sections of the eye showed moderate papilledema and numerous dilated preretinal veins. The intraocular blood noted grossly was confined by the internal limiting membrane to the retina; there was no extension into the vitreous chamber (fig. 6). One of the dilated preretinal veins located in the nerve fiber and ganglionic cell layers and in juxtaposition to the hemorrhage was clearly ruptured at a point where its wall had become necrotic (figs. 6 and 7). The vessel was obviously the origin of the preretinal hemorrhage. The central artery and vein were

somewhat dilated in the region of the optic disc. No dilatation of the choroidal vessels was observed.

Other microscopic sections confirmed the findings of pneumonia and the left cystic ovary.

Anatomic diagnosis. The following anatomic diagnosis summarizes the findings in the case: Ruptured congenital medial-defect aneurysm of the right internal carotid artery at its termination; massive subarachnoid hemorrhage, with hemorrhage in the optic nerve at its point of entry into the intraorbital space of the right eye; hemorrhage along the course of the right optic nerve in the subarachnoid and subdural spaces; rupture of a preretinal vein with associated preretinal hemorrhage in the right eye; right ventricular dilatation of the heart; anemia; hemorrhage, acute congestion, and early pneumonia of both lungs; left ovarian cyst; recent corpus luteum; abdominal adhesions; meningioma.

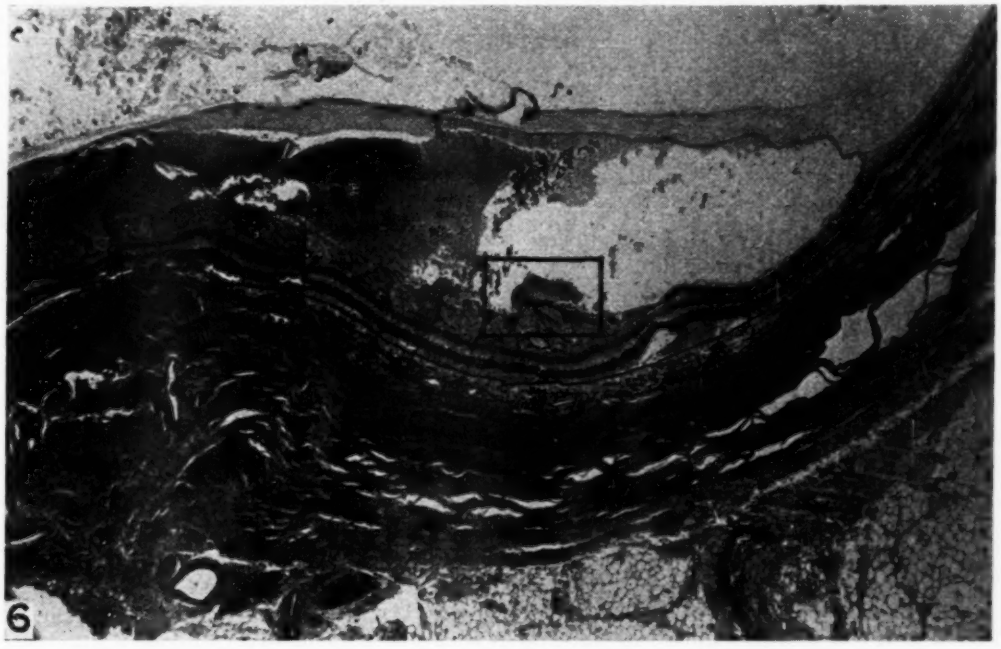

Fig. 6 (Miller and Cuttino). Preretinal hemorrhage at the site of rupture of the preretinal vein. The confinement of this hemorrhage by the internal limiting membrane of the retina is well shown. Hematoxylin and eosin (×35).

The dynamics of preretinal hemorrhage

To acquire a satisfactory understanding of the possible mechanisms responsible for preretinal hemorrhages a detailed knowledge of the vascular supply of the eyeball and optic nerve is essential. The ophthalmic artery, in supplying the eyeball, has two components; namely, the central artery of the retina, which supplies part of the optic nerve and all of the retina, and the ciliary artery, which supplies the remainder of the eyeball.[4, 5] Shortly after its entry into the proximal part of the optic nerve the central artery of the retina gives off a recurrent branch. In its intraneural course it supplies branches to the choroid at the level of the lamina cribrosa. These branches enter numerous capillary plexuses, through which they eventually anastomose with branches of the anterior ciliary artery in the region of the ciliary process. The central artery then continues to the optic disc, where it divides

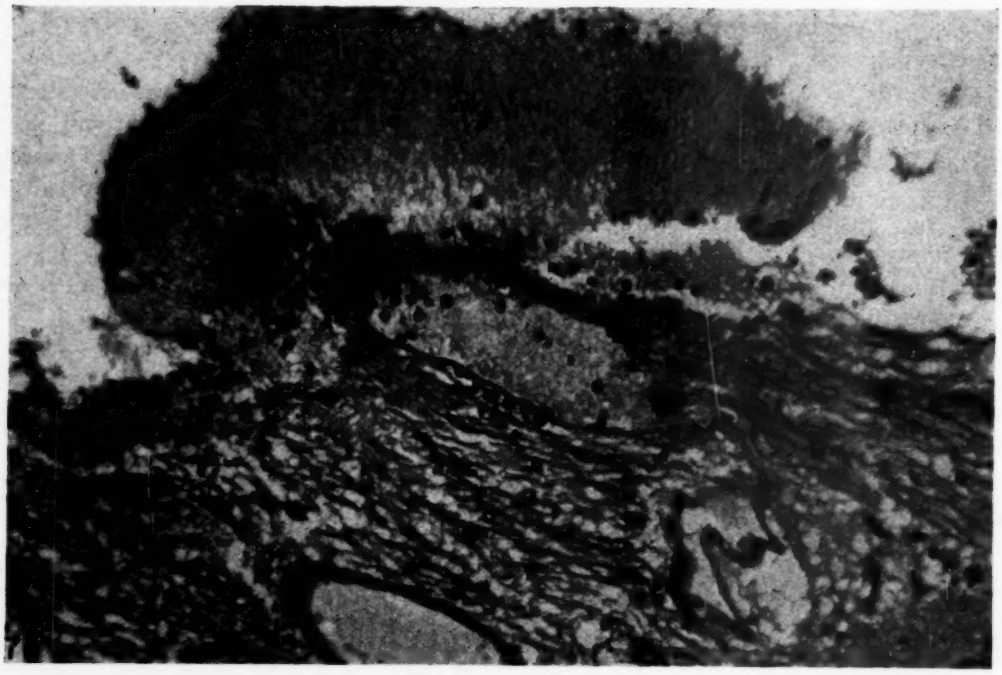

Fig. 7 (Miller and Cuttino). High-power view of the ruptured preretinal vein, as shown in bracketed zone of Figure 6. Half of this vessel wall shows necrosis and is the origin of the hemorrhage. Hematoxylin and eosin (×250).

into two branches, directed upward and downward, respectively. Each of these two primary branches divides into nasal and temporal secondary branches. Divisions of the latter follow a dichotomous pattern.[26] The vascular network into which the central artery ultimately breaks up in the retina has no connection with any other artery in the eyeball.[5, 26] Thus, it is generally agreed that the terminal branches of the central artery are end-arteries, analogous to the subpial arteries of the brain. These terminal branches lie against the inner layers of the retina; namely, the plexiform and ganglion-cell layers, from which position they extend outward to the inner nuclear layer.[27] The outer layers of the retina are nourished by the transudation of fluid from the choroid vessels.[5, 26]

The venous drainage of the retina parallels the arterial supply; there are no anastomotic connections of the veins in the retina with either those of the choroid and sclera or those of the ciliary body. These retinal veins all drain into the central vein at the optic disc. The latter, as it passes through the lamina cribrosa, has a limited anastomosis with the choroidal veins;[5, 26] except for this anastomosis, the central vein is considered an end-vein. The central artery and vein lie parallel and adjacent to each other in the center of the optic nerve for a distance of 5 to 20 mm. behind the eyeball.[8, 9] At this point the vein usually takes a position at right angles to the artery and reaches the periphery of the nerve nearer the eyeball than the point of entry of the artery into the nerve. Most commonly it runs for a variable distance in the subarachnoid space before transversing the intervaginal space and piercing the dura. The central artery, on the other hand, leaves the dura, crosses the intervaginal and subarachnoid spaces immediately and enters the nerve. Upon reaching the center of the nerve, it turns anteriorly, forming a 90° angle, and

takes its position adjacent to the vein. The central vein, thus, usually has a longer course in the subarachnoid space of the nerve than has the artery. The vein then proceeds proximally into the orbit to drain most commonly into the cavernous sinus.[5, 10] However, on at least one side anastomosis with the ophthalmic veins is present, thus making it possible for blood to drain either into the angular and facial veins or downward through the inferior orbital fissure into the pterygoid plexus.

The theories advanced to explain preretinal hemorrhages, like those utilized in the pathogenesis of papilledema, depend heavily upon the demonstration of the free communication between the subarachnoid and subdural spaces of the cranial cavity and the corresponding intervaginal or perineural spaces of the optic nerve through the optic foramen.[11] This anatomic relationship has been proved to exist by injection experiments in which various dyes were used[16, 28] and by study of the distribution of blood following spontaneous subarachnoid hemorrhage due to ruptured intracranial aneurysms;[1-3, 13-14, 19, 21,29] the extravasated blood has been traced from the cerebral subarachnoid space as far forward as the lamina cribrosa.

The earliest description of intraocular hemorrhage associated with spontaneous subarachnoid hemorrhage was contributed by Litten in 1881.[12] Symonds[1, 2] quoted a case, reported by Hale-White in 1895, in which the intracranial subarachnoid hemorrhage, extending into the intervaginal space of the optic nerve, had torn its way forward under the retina in both eyes. Symonds himself felt that there was sufficient pressure in the optic-nerve sheaths to force the passage of blood through the lamina cribrosa (fig. 1). Doubler and Marlow[13] described a similar case. Paton[14] thought that in a small percentage of cases retinal hemorrhages were due to direct leakage of blood from the optic nerve sheath through the lymphatic space at the site of the lamina cribrosa and along the central vessels into the optic disc and retinal tissues. He based his opinion on his failure to demonstrate dilated retinal vessels on ophthalmoscopic examination. In none of the more recent reports has continuity of the intervaginal hemorrhage with either the substance of the optic nerve or the retina been demonstrated.

Among the theoretical explanations proposed for papilledema, those based upon venous obstruction are of chief interest in conjunction with the pathogenesis of preretinal hemorrhages. Before the demonstration of the collateral circulation of the central vein in the orbit with the facial veins and the pterygoid plexus, it was thought that stasis of the ophthalmic vein resulting from obstruction of the cavernous sinus by thrombosis or compression from without was the cause of papilledema.[6, 11] Swift, however, has reconsidered this possibility recently in his discussion of anomalous and poorly developed anastomoses of the transverse sinus[15] and doubts this explanation.

According to Greear,[3] Uhthoff, in 1901, was the first to question the observation of direct hemorrhage from the intervaginal space of the optic nerve into the retina. Cushing and Bordley[16] were able to produce papilledema, as well as swelling of the intervaginal space, experimentally in dogs either by introducing fluid under tension into the intracranial subdural space or by simple digital compression exerted against dura exposed by a trephine opening. Their production of venous congestion by constriction of neck veins in the absence of fluid under tension in the perineural space failed to result in papilledema. Wolff and Davies[17] could not substantiate these results and pointed out that in dogs the central vessels do not cross the intervaginal space.

Paton and Holmes[7] in their study of the pathogenesis of papilledema concluded that it resulted from increased intravenous pressure which developed following compression of the intervaginal portion of the central vein. This compression was due to trans-

mitted increased intracranial pressure. In association with the increased pressure within the sheath of the optic nerve there was obstruction of the lymph drainage from the optic disc. Fry's[8, 9] studies on the anatomy of the central vessels supported their interpretation, as did the observation of Wallfoors (quoted by Lauber[18]), who demonstrated the continuity of the intracranial and intervaginal spaces by ligating the optic nerve proximal to the exit of the central retinal vein and thus preventing the development of papilledema when the intracranial pressure was increased.

Riddoch and Goulden[19] in interpreting their cases of intracranial aneurysm suggested that preretinal hemorrhages, as well as papilledema, could be explained on the basis of compression of the central retinal vein by the hemorrhage in the intervaginal space (fig. 2). The result of this compression was, according to these observers, an increase in the intraocular venous pressure, venous dilatation, and eventual rupture of the preretinal veins. They cited as variable factors the suddenness of rupture of the aneurysm, the distance of the aneurysm from the optic nerve, and the presence or absence of arachnoidal adhesions in the cisterna basalis. These variations tend to alter the degree of intervaginal tension and thus influence the production of preretinal hemorrhage. MacDonald[20] pointed out that the dura about the optic nerve in the orbit is the only portion of dura unsupported by a bony wall and therefore readily distensible. Drews and Minckler[21] affirmed the part played by increased pressure within the intervaginal space and suggested double angulation and axial elongation of the central retinal vein in its intervaginal course resulting from distension of the optic dura as additional factors responsible for the rise in central retinal venous pressure.

As a corollary, Igersheimer[22] has noted the absence of papilledema in a case with coëxistent glaucoma and intracranial neoplasm, the inference being that, in preexisting increased intraocular pressure, papilledema does not develop with increased intracranial pressure. Conversely, marked papilledema and retinal extravasation may develop under conditions of normal intracranial pressure when there is decreased intraocular tension.

Recently Ballantyne[23] has described hemorrhages occurring not only in the intervaginal space, but also in the optic nerve, the dural sheath, Tenon's capsule, the optic chiasm, the pial sheath in the sclera near the porus opticus, and in the orbital fat and muscle. He thought that the sudden rise in intracranial pressure at the moment of rupture of an intracranial aneurysm was followed by excessive venous stasis resulting from compression of the dural sinuses. Rupture of the distended veins in these scattered areas was, he thought, the result of "loci minori resistentiae." Because of the lack of continuity of the hemorrhages, he was not satisfied with the suggestion that the preretinal hemorrhage results from venous pressure due to simple extension of blood from the cerebral subarachnoid space into that of the optic nerve. It is the general consensus at the present time,[6, 11] however, that preretinal hemorrhages associated with extravasation of blood into the intervaginal space of the optic nerve, as well as those associated with papilledema, can be explained on the basis of obstruction of the central retinal vein as it passes through the intervaginal spaces of the nerve.

In our own case, described above, rupture of a congenital medial defect aneurysm of the right internal carotid artery has been demonstrated. Its histologic features are in accord with those described by Forbus.[30] Its location was such that blood could be traced into the subarachnoid space, through the optic foramen, and continuously along the intervaginal space of the optic nerve, terminating abruptly at the lamina cribrosa. In the eye an independent hemorrhage was found associated with a ruptured preretinal vein just beneath the internal

limiting membrane in the retina. Our interpretation of these findings is in general agreement with the consensus of opinion regarding the mechanism of production of preretinal hemorrhages; that is, that the hemorrhage is due to increased venous pressure. We think, however, that the *choroidal anastomosis* of the central vein at the lamina cribrosa, as well as the vein at its point of emergence from the nerve into the intervaginal space, is obstructed. Following rupture of the aneurysm the blood, under a high pressure simulating the systemic arterial pressure, is transmitted directly against the lamina cribrosa. The tissues of the lamina cribrosa are then compressed upon the *anastomosis* of the central retinal vein with the choroidal veins. The resulting sudden obstruction of the vein at its point of emergence from the nerve *and its choroidal anastomosis* is followed by rapid elevation of the preretinal intravenous pressure and rupture of a preretinal vein (fig. 3).

In support of this interpretation it should be recalled that the retina is supplied by end vessels and that their first anastomotic connections are with the choroidal vessels at the lamina cribrosa. Assuming the prevalent theory of obstruction of the central vein at its exit from the nerve to be correct, the result in that case would be a gradual compensatory distention of the choroidal anastomosis, and there would be no rupture of the distended preretinal veins. On the other hand, if the obstruction of the central vein occurred at its point of emergence from the nerve *and also at the choroidal anastomoses,* as suggested in our case, there would be no avenue of return for venous blood from the retina. The result would be an abruptly and markedly increased preretinal intravenous tension and almost immediate rupture of one or more preretinal veins. It might be added that the failure to demonstrate blood histologically in the intervaginal space would not be contrary to this view because pressure could be transmitted through spinal fluid trapped in perineural spaces.

Our interpretation is analogous to a theory postulated by Manz,[24] who thought that swelling of the optic disc results from compression of the lamina cribrosa by an increased cerebrospinal fluid pressure in the intervaginal space. It has further been shown in animals that ligation of the optic nerve distal to the exit of the central vessels from the nerve results in papilledema and swelling of the nerve distal to the point of ligation.[6] These findings were absent when the ligation was made proximal to the exit of the central vessels. They were particularly pronounced, however, when the ligation included only the vein. No mention is made of the simultaneous occurrence of preretinal hemorrhage in the experiments.

Summary

1. An instance of preretinal hemorrhage following massive subarachnoid hemorrhage from the rupture of a medial defect aneurysm of the internal carotid artery has been studied with particular reference to the pathogenesis of the ocular lesion.

2. Prevalent theories relating to the mechanism of production of preretinal hemorrhage accompanying subarachnoid hemorrhage have been reviewed and a modification proposed.

3. It is suggested that preretinal hemorrhages accompanying subarachnoid hemorrhage from ruptured medial defect aneurysms of the superficial cerebral arteries are the result of sudden increase in preretinal intravenous pressure which arises from compression of the choroidal anastomosis of the central vein of the retina at the level of the lamina cribrosa, together with compression of the central vein at its intervaginal portion, by extension of the subarachnoid hemorrhage into the intervaginal space about the optic nerve.

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THE SIGNIFICANCE OF THE BASE PRESSURE IN PRIMARY GLAUCOMA*

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The intraocular pressure varies throughout the day and from day to day in the normal as well as in the glaucomatous eye. Apparent variations may be due to the readings of two different tonometers, to a difference in the manner in which two individuals take the pressure readings, to changing muscle tension around the eyeball, to differences in the position of the tonometer foot plate on the cornea, and to the length of time the instrument rests on the cornea before the reading is made. Real variations may depend on whether or not miotics have been used, frequency of their use, and differences in strength, and the time elapsed since last used prior to the pressure readings. In glaucomatous eyes, variations in pressure no doubt depend on the current emotional state of the patient and the size of the pupil. Most important of all, however, are the real variations contingent on the glaucomatous state.

PEAK AND BASE PRESSURES

In glaucomatous eyes, the height to which the pressure rises is referred to in this paper as the "peak pressure" and the depth to which the pressure descends as the "base pressure." These terms represent merely the upper and lower limits of the excursions of the pressure taken from time to time. Any sustained elevation of the base pressure indicates permanent damage to the filtration angle. The difference between the base pressure and the peak pressure represents the transitory, functional factor in the glaucomatous state. The more advanced the glaucoma the more nearly the base pressure approaches the peak pressure, so that in absolute glaucoma they tend to merge.

The peak and base pressures are in a way comparable to the systolic and diastolic blood pressures. In both instances the lower pressure represents a rest period. A high diastolic pressure means less rest for the heart, and a sustained elevated base pressure means continuous damage to the eye with more rapidly developing field changes. A glaucomatous eye with a normal base pressure may for years show no field changes and no cupping of the disc but these changes may appear and progress rapidly when the base pressure is elevated. A further analogy to the heart can be made: in absolute glaucoma the base pressure tends to approach the peak pressure, and in a decompensated heart, the diastolic pressure approaches the systolic pressure.

STUDY PRESSURE TREND

For the purpose of this study the tension curves kept as a routine procedure in the office have been employed. This means that the intervals between pressure readings varied in length from a day to three or more months. Because of real and apparent variations already mentioned, frequent readings over a short period are not desirable. A long-range perspective, giving the general trend, is necessary. It is not so valuable to know that the pressure is 30 mm. Hg one week and 25 mm. Hg the next week as it is to know whether or not the reading of 25 mm. Hg is a sustained low or mean low, and whether or not it has risen to a mean 25 mm. Hg from a mean 15 mm. Hg. It is of more interest to compare the base pressure now with what it was a few months ago or even one year ago than to compare the current pressure with that of one week

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ago or even one month ago. Perhaps a more accurate comparison can be made between pressures taken at the same time of day. This is usually done, for patients generally pay visits to the office at about the same time. For our purposes, however, these small diurnal variations as well as the variations consequent to the changing emotional state, physical condition, treatment with miotics, and time elapsed since last medication can be disregarded in the long-range, over-all estimate of the pressure trend.

Classification of primary glaucoma

Fundamentally, I think the classification of primary glaucoma into shallow-angle and deep-angle glaucoma is sound.

A. Shallow-angle type

Shallow-angle or iris-block glaucoma is caused by some transitory dynamic factor back of the iris which pushes the root of the iris forward, blocking the angle. If this functional dynamic factor subsides or abates, the angle opens and the pressure returns to normal. If this blocking occurs often enough and over sufficiently protracted periods, the angle fails to open adequately because peripheral synechias form and the pressure is constantly elevated; that is, the base pressure has risen above normal. The functional episodes continue, so that the peak pressure soars, but in the interludes the pressure never returns to normal. Therefore, the height to which the base pressure rises represents the degree of permanent, irrevocable damage done the angle.

A common belief is that the functional factor is in the ciliary body in the nature of a vascular crisis. This produces edema of the ciliary body and processes which in turn push the iris root forward. There is not much histologic evidence to support this. In eyes approaching the absolute glaucomatous state when the base pressure nears the peak pressure, there may be evidence of increased vitreous pressure. This is frequently demonstrated on the operating table. If such eyes are palpated with an instrument such as a spatula, they will be quite hard even with the anterior chamber open. It seems possible, therefore, that transitory rises in vitreous pressure push the iris-lens diaphragm forward and block the angle.

B. Deep-angle type

The primary glaucoma, characterized by an angle which is of normal depth or deeper than normal, is thought to be due to trabecular obstruction. Not a great deal is known about the exact nature of the obstruction. Microscopic sections indicate that in some instances a cuticular product is laid down by the endothelium lining the interstices of the trabeculae, and in others there is a sclerosis of the trabeculae. The important point here for our discussion is that the changes leading to the glaucoma are commonly believed to be organic with no functional element. Were this true, the intraocular pressure should rise gradually with only small excursions due to variable factors not inherent in the glaucomatous state as previously discussed. Judging from a study of tension curves on this type of glaucoma, one can see that there may be excursions of pressure but never to the point of acute deep-angle glaucoma. This apparent variable functional element in this type of glaucoma is not readily explainable. It is true in general, however, that this type of glaucoma does show a more or less steady rise in the pressure, and that the apparent functional factor indicated by the excursion of pressure is an inconspicuous feature. Therefore, in this type of glaucoma we do not have the same occasion to speak of peak and base pressure as we do in the shallow-angle type. The pressure at any given time tends to represent the permanent organic damage to the angle.

In all glaucomas, primary as well as secondary, peripheral synechias eventually form. There is no exception to this, regardless of the causes of the glaucoma. Granting this, there is no escape from the fact that

prolonged increased intraocular pressure per se promotes the formation of peripheral synechias. Thus a vicious circle is formed by which "x," the unknown quantity initiating the glaucoma, causes an increase in the pressure which in turn promotes peripheral synechias which in turn produce increased pressure. This vicious circle, if continued, eventually causes complete annular peripheral synechia whereby the peak and base pressures merge and absolute glaucoma is reached.

Type of operation indicated

In determining the type of operation indicated, particularly in shallow-angle glaucoma where the functional feature is prominent, how high the pressure goes (peak) is not so important as how low it is capable of going (base). The higher the base pressure, the greater the drainage load the operation must carry. Sometimes it is impossible without several observations to determine the base pressure and therefore the operation indicated. For instance, if the intraocular pressure is found to be 35 mm. Hg even with miotics, it is important to know if this is peak, base, or average. If this is base pressure, then an operation capable of carrying a greater drainage load must be done than if the base pressure were 25 mm. Hg. Patients with glaucoma are too often sent to a consultant with instructions not to use miotic drops for 12 or more hours prior to the visit. If an opinion must be based on one visit, I should prefer to have the patient come with drops used routinely, since this gives a better idea of the base pressure. A pressure of 45 mm. Hg without drops is not as informative as a pressure of 20 mm. Hg with drops.

The difference between an acute glaucoma and an acute exacerbation of a chronic glaucoma is that in the former the base pressure is normal and in the latter it is elevated. This may be a difficult differential diagnosis to make but it is exceedingly important in determining the type of operation indicated.

A general working rule I employ for chronic primary glaucoma is:

A. Shallow-angle glaucoma

1. Low or normal base pressure—iridencleisis operation.

2. Rising base pressure to high normal, or up to approximately 30 mm. Hg—iridencleisis with sclerectomy or Lagrange type of operation.

3. Base pressure over 30 mm. Hg—trephining operation.

B. Deep-angle glaucoma

1. Base pressure of 30 to 35 mm. Hg—iridencleisis or cyclodialysis operation.

2. Base pressure of 35 to 40 mm. Hg—iridencleisis with sclerectomy or cyclodialysis operation.

3. Base pressure of over 40 mm. Hg—trephining operation.

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READING DIFFICULTY (DYSLEXIA) FROM THE OPHTHALMIC POINT OF VIEW*

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Reading difficulty, or dyslexia, is the result of the dysfunction of the various factors that are concerned in learning. Although the most prominent characteristic of dyslexia is an inability to read, the components of the problem are so diverse that any remedial program must be based on a thorough investigation of each individual reading failure. Not until the facts disclosed by the individual investigation are completely correlated and analyzed should any attempt either at diagnosis or prognosis be made.

Too often in the past, research studies in this field have been made to prove some preconceived idea that eventually influenced the research worker to interpret his findings and mold them into prejudiced patterns. The approach in previous investigations has depended largely on the particular field—psychology, education, ophthalmology, and so forth—in which the investigator was interested. An ophthalmic research worker would, for example, select a group of poor readers and attempt to correlate the ocular defects with the reading difficulties.

It is the purpose of this paper to present certain broad aspects of the problem of dyslexia, to outline the influence of certain physical factors involved in reading difficulties, to present some of the psychophysiologic elements, and to discuss the ocular findings in 133 cases of dyslexia under treatment.

BROAD ASPECTS OF DYSLEXIA

Patients with reading difficulty should not be neglected for fear that the difficulty might defy diagnosis and correction. Based on our results, it can be said that in otherwise normal children, dyslexia can be helped almost without exception. Although some patients, because of abnormal mental ability, do not show the expected improvement, most patients make gains that are both rapid and permanent. The most outstanding improvements in our experience have been a two years' gain in reading skill in two months; a five years' gain in 10 months; and a six years' gain in one year.

Since it would be unreasonable to expect a single individual, no matter how expert in his own field, to investigate and analyze reading failure on a sufficiently broad basis, it has been found necessary to coördinate the work among a group of experts. A staff capable of carrying on this work should include a skilled educational psychologist; teachers having special aptitude, interest, and experience in remedial instruction; a psychologist versed in psychometry and proficient in the art of interviewing and advising; a psychiatrist who understands the emotional development of the child as well as child-parent relationships; an internist; an otolaryngologist; an ophthalmologist; a social worker; and a voice teacher. This staff of experts must be willing to work together and to listen to each other's opinions.

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Before a course of treatment is prescribed, the patient's environment, physical status, emotional reactions, learning achievements, and work habits should be completely investigated. After each member of the staff has examined the patient, a conference should be held to report the findings and to establish the remedial program. It is advisable, even necessary, to consult with the patient's parents, siblings, and teachers in order to make recommendations and the reasons for them. Consulting with all persons concerned makes for better understanding. The patient, himself, should be given a clear explanation of the follow-up procedures.

Such a complex program can be facilitated if a central location can be provided to carry out the entire schedule. If a child should need not only medical care, including that of the eyes and ears, but also psychologic, educational, and psychiatric guidance, and voice training as well, the time consumed and the expenses incurred in getting treatments at different locations would make the prognosis less satisfactory.

Physical factors influencing dyslexia

Since vision is so dependent upon sufficient and proper lighting, one must determine what qualities and intensities of light are desirable for different reading situations. Much information is available concerning the optimum requirements for proper physical factors, which are controllable and necessary for comfortable reading.

Patterson and Tinker,[1, 2] and Luckiesch and Moss[3, 4] have investigated and reported extensively much valuable detail and information on the hygiene of reading. It has been estimated that the critical level for reading was between 3 and 4 foot-candles, but, as a margin of safety, reading should not be done with less than 5 foot-candles of light. School rooms should not have less than 10 foot-candles, and sight-saving classes should have 20 to 25 foot-candles. The critical level of light intensity for reading newspapers should be 7 foot-candles, but for a margin of safety, an intensity of 15 to 20 foot-candles is recommended. The critical level was defined as the intensity beyond which no improvement in reading performance occurred as the illumination intensity increased.

Maximum readability occurs when optimal physical conditions are supplied, with the printed material set in 10- to 12-point black type on a 2-point lead, in line lengths of 3 to 3½ inches, on white paper without gloss finish. Under these conditions, the light should not be less than 10 foot-candles. This light intensity should be increased even to 50 foot-candles for prolonged tasks of reading difficult material or for proofreading. Glares should be avoided at all times.

Pyscho-physiologic aspects

As a rule, the more intelligent a person is and the more perceptual experiences he has encountered, the more quickly he learns from abstractions and symbols. Since printed letters and words are abstract symbols, it is necessary to have visual perceptual experiences in order to understand their meanings when arranged in phrases and sentences.

If we accept the customary concept that reading is a simple reaction to visual stimuli, we would have to conclude that the visual process is the dominate factor in dyslexia. However, in order to analyze and interpret reading achievements and failures, it is necessary to consider the central perceptual and conceptual processes as well as the entire peripheral ocular mechanism in all its complexity.

There are many individuals with normal or superior intelligence who, although they are unable to understand printed signs and symbols, are able to learn through oral instruction.

Such a complex achievement as reading involves many variables, and it would be unreasonable to assume that any function can be altered from normal development

without influencing the entire psychophysiologic mechanism. The degree to which normal or abnormal functions influence the process as a whole depends upon the ability of the person to adjust himself to the psychophysiologic needs or contingencies or stimuli that are present at any particular time. The degree of this homeostatic[5] stability and adaptation depends on the relative degree of normality of all the diverse functions that are involved in the reading processes.

According to conservative estimates, about 15 percent of the school population has some degree of dyslexia. This varies considerably with the standard of the school and the opportunities of the home. The social-economic status of the family seems to have no significant influence on the development of dyslexia. In the series of cases presented in this study, the average age of the patient was 12 years and 2 months. Boys having reading difficulties outnumbered girls, 4 to 1. The average intelligence quotient in the patients herein studied was 107.6, with the highest, full-scale I.Q. at 147.

Reading readiness in general is a further consideration. The child's readiness for reading requires broad interests. He must have a certain degree of emotional and social maturity, a normal expectancy in language development, and a sufficient number of perceptual and informational experiences.

Wilson[6] found that letter-perception abilities are more closely related to beginning reading progress than any of the other abilities studied. Since so much depends upon accurate visual perception, it is essential to determine at what age a child's eyes are sufficiently mature for him to learn to read. It is no more reasonable to assume that every child will have fully matured visual development when he enters school at the age of six years than to say that all children are equally developed in other maturational processes. Park and Burri[7] have shown that a larger percentage of children at the prereading age and in the first and second grades have more incompletely matured visual mechanisms than children in the next higher grades.

If this slow development of "eye readiness" should persist, it would become a definite deterrent to reading readiness and eventually, because the criticisms of teachers and parents would arouse anxiety in the child, it might lead to nervousness, irritability, resentments, and behavior problems. Under these circumstances, it is advisable to postpone, or at least to modify the entire reading program until such immature maturational processes have been corrected.

Ocular study of dyslexia

Many reports have been made and many conclusions reached relative to the etiologic importance of ophthalmic findings in reading difficulties, some with finality, others with less certainty.

The following data were catalogued after an analysis was made of the ophthalmic factors in this study of 133 cases of dyslexia. The results and conclusions are presented with the preface that all data should be interpreted as being only a part of the complex problem and that the whole makeup of the child must be considered.

Visual examination

In this series, 75 percent of the patients had normal vision; 20 percent had subnormal vision in both eyes; and 5 percent had subnormal vision in one eye only. At the initial examination an additional 8 percent seemed to have subnormal vision, but these examinees attained normal vision easily at a subsequent examination. Perhaps, their lack of interest and attention in any task concerned with reading was carried over to the examination.

Only 19 percent showed sufficient ametropia to require refraction. There were twice as many hyperopic as myopic cases. Glasses were removed from six of our pa-

tients, five of whom were wearing spectacles because they had been prescribed empirically as an aid in reading. Three of these patients had a high degree of exophoria which was aggravated by the convex lenses. An inaccurate refraction influences reading adversely and is worse than ametropia itself, for the patient then has to compensate for an artificial ametropia.

The incidence of subnormal vision cannot be ignored as a factor in dyslexia but it is relatively rare. No doubt, to attain clear vision, ametropia with its accompanying excessive demand upon the ocular neuromuscular mechanism results in fatigue and would be a very definite factor in some cases. For as Dearborn[8] mentions, the fatigue would thus lessen the incentive to learn, causing avoidance or neglect of reading.

Oculomotor functions

Ninety-three percent of the cases in this series had normal motility during fixations into the various fields of action of the extraocular muscles both monocularly and binocularly.

Since fusion ability was present in practically all cases of dyslexia (97 percent), fusion per se is not a dominating influence. It is the ability to maintain fusion without undue effort and fatigue which determines the proper oculomotor function. Although fusion and stereopsis are acquired oculocerebral processes, 22 percent of our cases showed extremely weak stereopsis or none at all. Of these, only 4 percent were due to tropias. The remaining 18 percent learned stereopsis properly after training. The question naturally arises whether or not weak stereopsis is another manifestation of the dyslexia enigma.

Ocular dominance

The number of investigations dealing with the controversial question of dominance, handedness, eyedness, have been considerably smaller during the last 5 years than the preceding 10 years. Although the results are still somewhat conflicting, the evidence points to the conclusion that handedness is not significantly related to reading ability but that mixed eye-hand dominance is probably present in some cases of reading disability.

In checking for ocular dominance, 80 percent of our cases showed no preference; 8 percent had a tendency to alternate; 2 percent had a tendency to alternate but recovered quickly; 3 percent showed a tendency to suppress one eye; and 7 percent suppressed one eye completely. By using the stereoscope with dissimilar cards that stimulate fusion only slightly, the probability of introducing an extraneous factor in determining the dominance is eliminated. For, as soon as an extraneous act such as pointing and sighting is used, one eye or the other, through expediency, will be chosen as the dominant one, even though both eyes are maintaining fixation and fusion of the image of the object observed. Apparently not more than 13 percent of dyslexia cases show any tendency to fixate alternately or to suppress one image, unless this suppression is due to a strabismus.

Incidence of phorias

Phorias have more significance, for 55 percent of the patients were orthophoric. The remaining 45 percent were heterophoric, and half of them were exophoric for near (reading distance). This condition with its potential of temporary diplopia can be a definite hindrance to the proper functioning of the eyes, causing a predisposition to fatigue and eventual lack of interest in reading or doing other tasks close at hand. The Maddox rod and red light were used to measure phorias. The various types of phorias present and their relative incidences were:

Orthophoria, 55 percent; esophoria for distance and near, 5 percent; esophoria for distance only, 2 percent; esophoria for near, 4 percent; exophoria for distance and near, 4 percent; exophoria for distance only,

2 percent; exophoria for near, 22 percent; esophoria for distance and exophoria for near, 2 percent; hyperphoria, 4 percent; tropia, 4 percent. From 4 degrees esophoria to 4 degrees exophoria was considered as orthophoria; less than 2 degrees was normal for hyperphoria.

Measurement of ductions

During the measurements of ductions, care was taken with each individual to obtain accurate reports as to the point at which the image separated and diplopia appeared and the point of recovery—where the two images fused again. In scoring for ductions, the standards were quite flexible. A variation from the established normal of approximately 10 percent was allowed as "normal." The phorometer was used to make these measurements which are analyzed in Table 1.

TABLE 1

An analysis of ductions

	Diplopia Considered Normal	Recovery from Diplopia Considered Normal	Percentage of Entire Series (133 Cases)		
			Lower Ability	"Normal"	More than Normal Ability
			Percent	Percent	Percent
Distance					
Adduction	18*		17	66	17
Adduction		10*	28	55	17
Abduction	8†		20	74	6
Abduction		5†	25	70	5
Near					
Adduction	24*		15	60	25
Adduction		13*	22	61	17
Abduction	14†		10	63	27
Abduction		6†	15	57	28

* Prism base out.
† Prism base in.

Even with a relatively low prism strength considered as normal in measuring the diplopia and recovery points, it is evident that weak fusion amplitude is definitely associated with dyslexia, especially weak recovery ability from diplopia. With a low duction reserve in the presence of convergence insufficiency or accompanying accommodative or convergence spasm, there is a tendency toward undue fatigue that prevents the eyes from efficiently making the fusional responses and adjustments so necessary in getting and maintaining single and clear binocular vision. If this is not accomplished efficiently, there is a constant tendency for the two images to fail to remain on the fovea and, as a result, there is blurring of the images or even potential temporary diplopia. An unusual amount of nervous energy is required to adjust the foveal positions constantly in order to achieve and keep a single image. This may be evidenced by some general physical and emotional disturbance and can happen even though the vision may be normal or may have been improved to normal by refraction. Thus, in studying the correlation of the eyes to dyslexia the entire peripheral ocular mechanism must be considered.

Orthoptic treatments were recommended in 69 (52 percent) of our cases showing abnormal ductions or marked muscular imbalances. Eleven of these were orthophoric, 5 had tropias (3 of which were of such magnitude it was impossible to measure their ductions), and the remainder were heterophoric.

Observations were made by motion pictures* of the eyes while reading. This included rate of reading, average fixations calculated per 100 words, span of recognition, number of regressions per 100 words, saccadic movements, whether rhythmic or arrhythmic, whether eyes are synchronized, and so forth. The results are indicated in Table 2.

grade material is attributed to the circumstance that even though their average reading ability was on the 2.12 grade level, they refused to try any reading on that level. In the over-all picture of the peripheral ocular function while reading on the other grade levels, the dyslexia cases are inferior to the "norms."

Exercises were given to 15 percent of our cases to stimulate rhythmic saccadic movements, to increase the span of recognition, to overcome regressions, and to regulate the ocular speed in reading.

TABLE 2

ANALYSIS OF OCULAR MOVEMENTS IN READING

Grade level used to photograph eye movements	1	2	3	4	5 to 7	High School and College
Reading ability from psychologic report (average)	2.12	2.8	2.8	4.1	5.9	10.3
Rate per minute						
Norms*	55	90	115	168	200	310
Dyslexia	83	66	97	95	161	250
Fixations per 100 words						
Norms*	190	150	137	115	100	70
Dyslexia	164	181	162	181	119	96
Regressions per 100 words						
Norms*	40	32	30	25	17	13
Dyslexia	19	43	29	35	23	12
Span of recognition Norms	.52	.66	.73	.87	1.0	1.4
Span of recognition Dyslexia	.61	.55	.61	.55	.84	1.0

* Taylor, E. A.: Controlled Reading. The University of Chicago Press, 1937.

There were several patients for whom no reading graphs were made for they lacked the ability to read even first-grade material.

The excellent showing made by the group that was photographed while reading first-

It would be erroneous to take the peripheral ocular mechanism habits as the standard and the visual acuity as the determinant of cerebral action or of perceptual processes. It is also inconclusive to take mental processes as the sole factor influencing the modus operandi of peripheral ocular performance. Since the roles are probably interchangeable, it would be difficult to determine which acts as the originator and which as the compensator. All available faculties plus the desire to achieve the universal concept are probably integrated in the processes of learning.

* Four types of eyes—emmetropic, hyperopic, myopic, and aphakic—were also photographed.[9] Conditions under which these patients read were identical, except for their refractive corrections. The results showed that the excursion of the eyes in reading a line was influenced by the power of lenses used in myopia and aphakia. The excursions of emmetropic eyes and hyperopic eyes (with +11D. to +13D. lenses) averaged 6.5 to 7 mm. The excursions of aphakic eyes reading under similar conditions (with +13D. lenses) were increased even to 10.5 or 11 mm. The myopic eyes moved approximately 5 to 5.5 mm. This occurs in individuals who have had no difficulty in reading and helps explain the marked difficulty which some aphakic patients have in adjusting to lenses, especially while reading.

Conclusions and Summary

1. Dyslexia is an entity.

2. The incidence of dyslexia is sufficiently high to require attention.

3. The correction of this condition can be best attained after a complete examination and appraisal of the child and his problem from every viewpoint, including physical, emotional, and pedagogic.

4. A somewhat detailed analysis is made of the ophthalmic findings that were coincidental with other symptoms in a series of 133 cases of dyslexia.

5. Ametropia must be considered as a factor in some cases of dyslexia.

6. Refraction should correct the ametropia but the empirical prescribing of glasses to help a child overcome reading disabilities is not advisable.

7. Abnormal motility is rare in dyslexia cases—probably as rare as in an unselected group.

8. Fusion is present in practically all cases, so fusion per se seems not to be a dominant factor.

9. The incidence of the lack of stereopsis is rather high.

10. Phorias are significant, especially exophoria for reading distance.

11. Weak ductions, slow recovery ability after diplopia, heterophoria, convergence insufficiency or accommodative or convergence spasm are associated with half of our cases.

12. Orthoptic treatments were recommended in 52 percent of our cases.

13. Exercises were given in 15 percent of our series to stimulate rhythmic saccadic movements, to increase span of recognition, to overcome regressions, and to regulate the ocular speed in reading.

14. It is erroneous to take the peripheral ocular mechanisms as determinants of perceptual and conceptual processes.

15. Conversely, it is illogical to take the cerebral processes as determinants of the ocular functions.

16. The roles of originators and compensators are probably interchangeable when an abnormality is present. Otherwise, there is perfect homeostasis between these sensory and motor functions.

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THE SIGNIFICANCE OF THE INTRACUTANEOUS TEST FOR HYPERSENSITIVITY TO UVEAL PIGMENT*

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The intracutaneous test for hypersensitivity to uveal pigment is the direct outgrowth of Elschnig's old anaphylactic theory of sympathetic ophthalmia.[1] The test was first used clinically by one of us (A. C. W.) in 1921.[2] At first it was read on a clinical basis, based on erythema and induration at the end of 30 minutes, 24 hours, and 48 hours. In 1934 Friedenwald studied the histologic changes accompanying a positive test.[3] He showed that two weeks after the injection of the pigment into the skin of a hypersensitive individual, there occurred in the skin a remarkable and typical histologic picture, characterized by phagocytosis of the injected pigment, infiltration of the dermis with lymphocytes, giant cells, and epithelioid cells, the latter usually occurring in nodules. The picture in the skin was startlingly similar to the picture of sympathetic ophthalmia in the eye.

Since the publication of Friedenwald's paper, chief reliance has been placed on the histologic reading of the excised skin. This test has been used extensively in the Wilmer Institute since 1934. It is the purpose of this report to summarize our experiences with this test, to discuss its occurrence in various ocular conditions, and to attempt an evaluation of its significance, its diagnostic and its prognostic import.

TECHNIQUE

A. PREPARATION OF PIGMENT

The uveal pigment is prepared from fresh beef eyes obtained from the slaughter house. Under sterile precautions, the eyes are clipped clean of the surrounding orbital tissue. They are then dipped for a few seconds in boiling water and further cleansed. The corneas are then removed, a sagittal section made in the eye, the vitreous, lens, and retina removed. The uveal tract is then dissected out intact and clipped free at the optic nerve. The uveal tracts of all the eyes thus dissected are ground up in a sterile mortar with glass, sand, and salt solution. The pigment is thus extracted from the uveas and the pigment-containing solution poured off and fresh salt solution added until the supernatant fluid becomes pigment free. The pigment-containing salt solution is then diluted so the end dilution is 5 cc. of solution to each uveal tract. This gives almost a 0.5-percent (wet weight) concentration of pigment, together with the soluble protein extracted from the uveal tract. Tricresol is then added to 0.5 percent. The mixture is then agitated several hours in a shaking machine with glass beads. It is then incubated for 48 hours, and its sterility tested. Thereafter it is decanted under sterile conditions and stored in treatment bottles for use.

B. TECHNIQUE OF TEST

An injection of 0.1 cc. of the pigment solution is made intracutaneously in the forearm. As controls, 0.1 cc. of salt solution, containing 0.5-percent tricresol, and preferably 0.5-percent beef serum, is injected as a control. The test is read at 30 minutes, at 24 hours, and at 48 hours clinically. It is then left for 14 days at the end of which time the pigment-containing skin is excised and prepared for microscopic study.

C. RESULTS

The tests are reported here on the microscopic reading only. They are read as positive, doubtful positive, negative, nonspecific

* From the Wilmer Ophthalmological Institute of The Johns Hopkins University and Hospital.

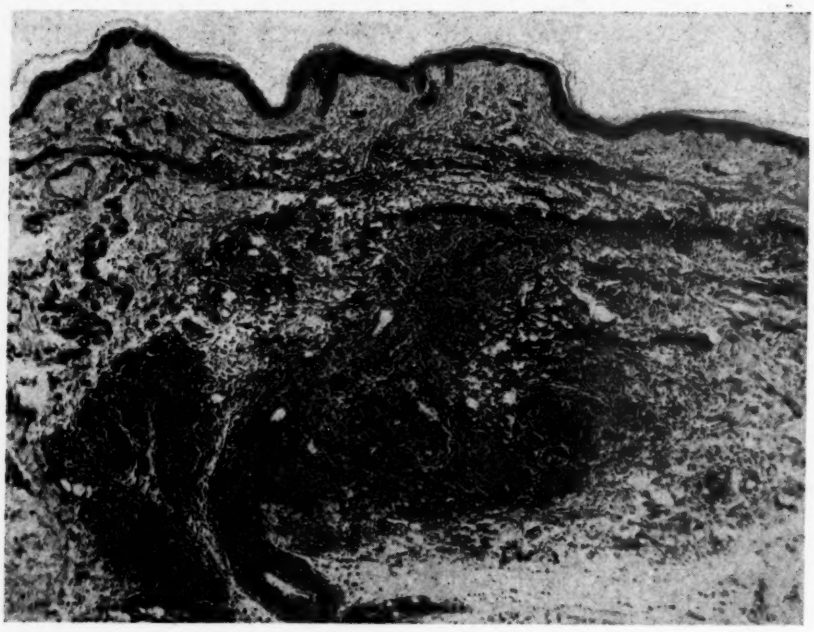

Fig. 1 (McPherson and Woods). Positive reaction.

reaction, or infected. The following are the criteria for the various readings:

Positive Test. All of the pigment is phagocytosed by the epithelioid and giant cells. The area of the section is densely infiltrated with lymphocytes, epithelioids, and giant cells, which are evenly dispersed throughout the section and (fig. 1) in some places form nodules—giant cells circumscribed by epithelioid cells.

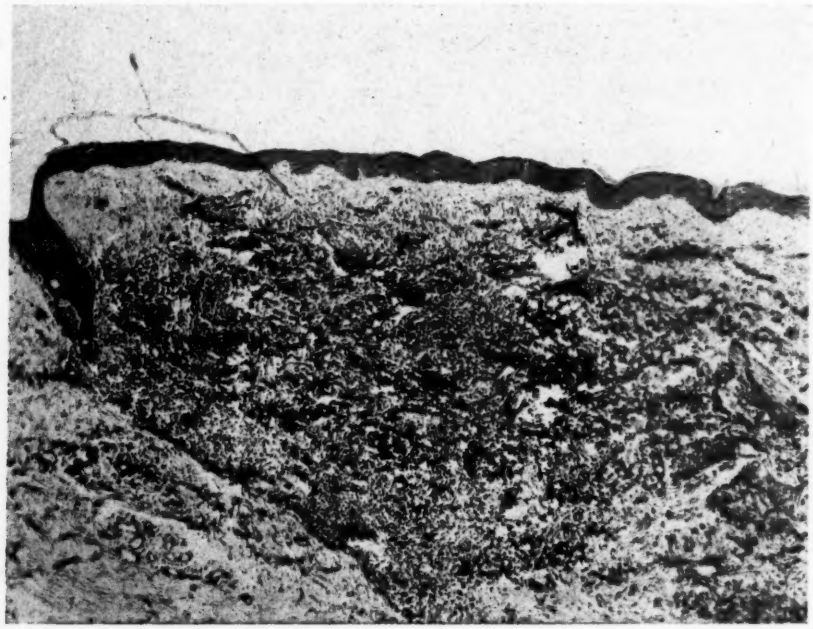

Fig. 2 (McPherson and Woods). Doubtful-positive reaction.

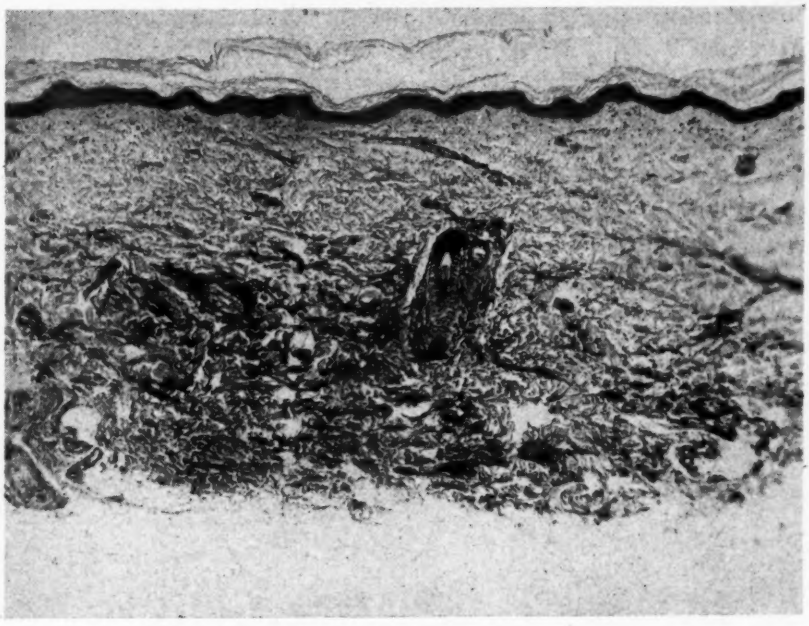

Fig. 3 (McPherson and Woods). Negative reaction.

Doubtful Positive. A portion only of the pigment is phagocytosed by the epithelioid and giant cells, the remainder being inertly distributed throughout the dermis. The cellular reaction of lymphocytes, epithelioid and giant cells is less intense than in the positive reactions, and is spotty in its distribution. There is no typical nodule formation (fig. 2).

Negative Reaction. All the pigment lies inertly distributed in the dermis, usually in large clumps. There is no phagocytosis and

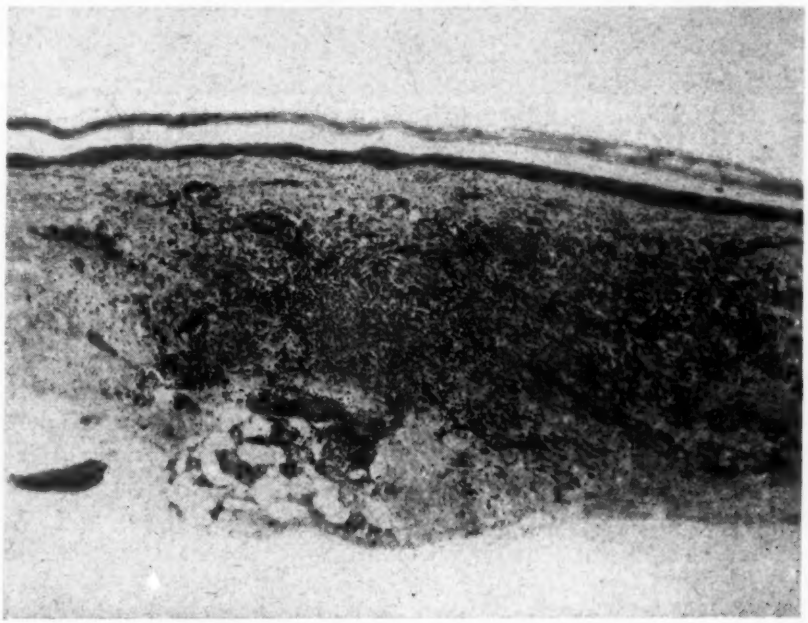

Fig. 4 (McPherson and Woods). Nonspecific reaction.

no cellular reaction other than an occasional lymphocyte (fig. 3).

Nonspecific Reaction. The greater portion of the pigment lies inertly distributed in the dermis. There is a moderate cellular reaction chiefly of lymphocytes and wandering mononuclear cells. There may be an occasional epithelioid cell containing a little phagocytosed pigment, but such cells are extremely scanty (fig. 4).

Infected. The pigment is largely unphagocytosed in the dermis. The entire section is infiltrated with polymorphonuclear leukocytes, which occasionally phagocytose some of the pigment. There is often severe necrosis (fig. 5).

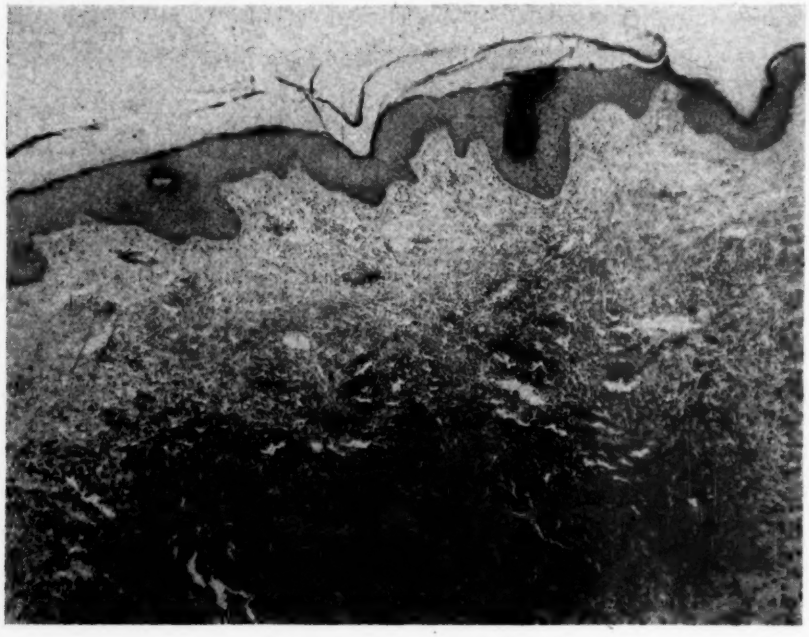

Fig. 5 (McPherson and Woods). Infected.

Material

The material on which this study is based is (1) 30 cases of sympathetic ophthalmia; (2) 139 cases of intraocular operations or injuries involving the uveal tract, in some cases healing uneventfully, in others complicated by a postoperative or posttraumatic uveitis; and (3) 43 cases of endogenous uveal disease from constitutional causes without a history of injury. In many of the patients there were repeated tests available for study. The following are the results in these three groups.

Results

I. Sympathetic ophthalmia

There are 30 individual cases in this group. In 22 of these, the diagnosis was histologically proven by examination of the exciting eye. In eight patients the diagnosis was unproven, the reasons being either that the exciting eye had never been enucleated and there was no reason for enucleation in the stage in which the patient was first seen, or the eye had been enucleated elsewhere and the sections were unobtainable for study. However, in none of these cases did there appear any reasonable doubt of the diagnosis. Therefore, both the 22 histologically proven and the 8 clinically diagnosed cases are grouped together.

The results of the pigment tests in these

TABLE 1

RESULTS OF PIGMENT TESTS IN 30 CASES OF SYMPATHETIC OPHTHALMIA

Individual	Hospital Number	1st	2nd	3rd	4th	5th	6–8	9–12	1 yr. +	2 yrs. +	Remarks
W.A.	204638			neg.	++++ pos.		++++ pos.		neg.		
L.B.	409440				++ doubt.						
R.B.	384219			++ doubt.							never retested
H.B.	327584		++++	++++							
M.B.	124865	++++									
A.B.	217703	++									never retested
M.R.C.	207520	++++	neg.								
M.G.C.	170244	neg.									never retested
A.S.G.	305525			neg.							never retested
M.H.	155979			++++		++++	++++	++++			
S.J.H.	313990				++++						
B.K.	191216							++++	++++	++++	positive 2 yrs. 4 mos. after onset—still active
H.L.	375439								neg.		active 18 mos. after onset, and was negative—no repeat
M.M.	397715	neg.		neg.							never retested after 3rd mo.
S.M.	104603			++++ ++++	++			++++		neg.	inactive at end of 2 yrs.
J.P.	307122		++								never retested
H.P.	372462	neg.		neg.							never retested
P.L.R.	250487						++				inactive after 18 mos.
H.R.	072199	neg. ++++									negative on 14th day —positive on 30th day
E.T.	410364	++++									
H.V.	113134	++									never retested
M.W.	247541	++++									
A.A.	U.75495						++++				
O.B.	249495	++++									
F.D.	259772								++	neg.	
E.J.	107915	++++									
E.S.	24138	(5) ++++									5 positives in 1st mo. —2 yrs. "necrotic"
W.S.	U.75341									++++	1st attack sympathetic 1913—onset 20 yrs.—seen first 2 yrs. after exacerbation—still active
L.S.	100802						++++ ++++		++ neg.		negative after 16 mos.
M.S.	135106			++++ ++++							

25 positives and doubtful positives = (18 positives—7 doubtful) = 83%.
5 negatives—tested = 4 tested in first 3 mos. and never retested. 1 tested 18 mos. after onset—still active.

30 patients are given in Table 1. Eighteen of these 30 cases, or 60 percent, gave straight positive reaction, while seven, or 23 percent, gave doubtful, or weakly positive reactions. Of these 25 strongly or weakly positive reactions, 10 occurred in the first month of the disease, while four patients tested in the first month were negative. Two patients were examined for the first time in the second month of the disease, both being positive. Four patients were tested for the first time in the fourth month of their disease, and all four tests were positive. The remaining four straight positive reactions were found in patients tested for the first time anywhere from the fourth month to the second year after the onset of the disease. One patient who gave a negative result on the first examination in the third month of the disease became positive in the fourth month. Of the five negative tests, four were done in the first three months of the disease, and the patients were not tested thereafter. The one remaining negative patient was first tested at the end of 18 months after the onset of symptoms.

This evidence would indicate that the tendency, if not the rule, is that the test becomes positive by the fourth month after the onset of symptoms, and in the majority of cases, becomes positive within the first month. If this is true, as it appears to be, the fact that the test was negative in five patients must be explained by the time at

which the test was done. Thus in four of the five negative patients the tests were done in the first three months. Had they been repeated later, positive results might have been obtained. The fifth case was tested 18 months after the onset of the disease, when the sensitivity may have faded, there being several cases in the positive group which were positive in the early stages of the disease and became negative by the end of the first year.

On the strength of this evidence—that 25, or 83 percent, of the cases gave strongly or weakly positive reactions to pigment while the five negative cases occurred in stages where collateral evidence indicates a negative test might well occur—it seems a reasonable deduction that hypersensitivity to uveal pigment is a fairly regular concomitant of sympathetic ophthalmia.

TABLE 2

PIGMENT REACTIONS IN TRAUMATISM OF UVEAL TRACT, POSTTRAUMATIC OR POSTOPERATIVE COMPLICATIONS

Type of Case	No.	Positive ++++	Doubtful Positive ++	Negative	Non-specific	Infected
Penetrating wounds of Uveal Tract—Enucleation of Injured Eye	37	3	3	29	1	1
Recurrent Postoperative Uveitis	37	7	7	18	1	4
Postoperative Infection or Hemorrhage	20	3	2	15	0	0
Totals	94 or 100%	13 or 14%	12 or 13%	62 or 66%	2 or 2%	5 or 5%

II. PIGMENT HYPERSENSITIVITY IN INJURIES AND OPERATIONS INVOLVING THE UVEAL TRACT

If it is granted that hypersensitivity to uveal pigment is part of the disease picture of sympathetic ophthalmia, the immediate question is whether or not a hypersensitivity to pigment occurring after an operation or trauma to the uveal tract, but without clinical evidence of sympathetic ophthalmia, should be regarded as an indication or harbinger of impending sympathetic disease. This question can be answered only by an over-all study of the results of pigment tests in patients with injuries or operations involving the uveal tract, and a comparison of the results of these pigment tests with the subsequent clinical course of the patients.

There are a total of 139 cases of various penetrating wounds of the eye involving the uveal tract and intraocular operations involving the uveal tract in which, for one reason or another, tests for pigment hypersensitivity were done. None of these patients had or later developed sympathetic ophthalmia. This material falls into two groups. *Group A.* Patients who ultimately lost their eyes as a result of the injury or operation, or who suffered postoperative uveitis, infection, or hemorrhage. There were a total of 94 patients in this group. *Group B.* Patients in whom the course after injury or operation was uneventful and who healed without complication. There were a total of 45 patients in this group.

Group A. The results of the test for pigment hypersensitivity in this group are shown in Table 2. In 13 patients, or 14 percent, the tests were strongly positive. In 12, or 13 percent, the tests were doubtful positive. In 62, or 66 percent, the tests were entirely negative. In seven patients the test was either nonspecific or infected.

Group B. The results of the test for pigment hypersensitivity in this group are shown in Table 3. No positive tests were found. Seven, or 16 percent, showed doubtful positive reactions. The remaining 38, or 84 percent, were entirely negative.

TABLE 3

PIGMENT REACTIONS IN TRAUMATISM OF UVEAL TRACT—UNEVENTFUL RECOVERY

Type of Case	No.	Positive	Doubtful Positive	Negative	Non-specific	Infected
Penetrating Wounds of Eye—Uneventful Recovery	38	0	3	35	0	0
Operations on Uveal Tract—Uneventful Recovery	7	0	4	3	0	0
Totals	45	0	7 or 16%	38 or 84%	0	0

Comparing these two groups it is at once evident that the occurrence of a strongly positive pigment test after an injury or operation involving the uveal tract is a harbinger or a warning of a stormy postoperative course, but it is not an indication that sympathetic ophthalmia will develop.

Doubtfully positive and negative tests are manifestly of no prognostic significance, occurring in much the same proportion in postoperative or posttraumatic cases, regardless of whether there are posttraumatic or postoperative complications or uneventful healing.

III. Pigment hypersensitivity in endogenous uveal disease

There are 42 patients in this category on whom pigment tests were done and the injected skin studied histologically. These cases fall into two distinct groups. In the first group are 37 cases of endogenous uveal disease from various constitutional causes, and in the second group five cases of the amazing Vogt-Koyanagi syndrome—uveitis with dysacousia, poliosis, vitiligo, and alopecia. The results in these two groups are shown in Table 4.

In the 37 patients with ordinary uveitis from constitutional causes, there were no positive tests. Doubtful positive tests occurred in five patients and negative or nonspecific in 32 patients. The notable finding is the complete absence of positive tests. The doubtful cases are difficult to evaluate. They suggest that occasionally some incomplete immunologic reaction involving pigment may occur. It is interesting that the disease was bilateral in four of these five cases with endogenous uveitis and doubtfully positive tests. The possibility of some tissue reaction in bilateral uveitis might be explored.

In the Vogt-Koyanagi syndrome, the test was positive in three of the five patients tested. The etiology of this disease complex is unknown. All that can be said is that clinically and histologically the ocular picture of the disease resembles sympathetic ophthalmia.

Discussion

In the cases which form the basis for this study, clear-cut positive skin tests for pigment hypersensitivity occurred only in three conditions: (1) in sympathetic ophthalmia; (2) in the prolonged uveitis after injuries or

TABLE 4

PIGMENT TESTS IN ENDOGENOUS UVEAL DISEASE

Type of Cases	No.	Positive	Doubtful Positive	Negative	Non-specific	Infected
Uveitis from Constitutional Causes	37	0	5	30	2	0
Vogt-Koyanagi Syndrome (Uveitis—dysacousia, poliosis, vitiligo, alopecia)	5	3	0	2	0	0

operations involving the uveal tract; (3) in the Vogt-Koyanagi syndrome.

The mechanism of these positive tests can be well understood. It has been clearly shown that uveal pigment possesses organ-specific and lacks species-specific immunologic properties, although it has not been shown whether this is an attribute of the matrix of the pigment-containing cells or of the melanin granules. The relative insolubility of the melanin, with some unpublished experimental work in the Wilmer Institute, suggests the first possibility. Thus theoretically, and indeed experimentally, the pigment (using the term in the sense of pigment-containing cells) can act as an antigen in the homologous animal. When pigment hypersensitivity occurs after an ocular injury or disease, the obvious explanation is that uveal pigment is absorbed from the eye, the cells of the body react to the pigment, and a generalized hypersensitivity ensues. In sympathetic ophthalmia and in the Vogt-Koyanagi syndrome further absorption of pigment from the eye results in a local hypersensitive reaction in the eye manifested by phagocytosis of the pigment; epithelioid, giant cell, and lymphocytic cellular infiltration; and nodule formation. Likewise, a similar reaction occurs when the pigment is introduced artificially in the skin.

In the cases with persistent posttraumatic or postoperative uveitis which show positive skin tests, the mechanism of sensitization is the same; but when the process stops, sympathetic ophthalmia does not occur. The eye shows only the picture of nonspecific endophthalmitis. The obvious explanation for this is that after the establishment of the pigment hypersensitivity by the initial absorption of pigment, there is no further absorption from the eye, and hence no local hypersensitive reaction. When the hypersensitive skin is injected with uveal pigment, it reacts in the classic manner.

Does the occurrence of hypersensitivity to uveal pigment in sympathetic ophthalmia throw any light on the riddle of the etiology of the disease? In consideration (a) of the fact that pigment hypersensitivity may develop after ocular injury and without sympathetic ophthalmia and (b) of the identical histologic picture of pigment hypersensitivity in the skin and sympathetic ophthalmia, in 1935 one of us (A. C. W.) advanced the following hypothesis. Pigment hypersensitivity is not the actual cause of sympathetic ophthalmia, but it is a prerequisite for its development. It sets the stage for the occurrence of the disease, and determines the histologic pattern. Some other unknown factor, possibly specific, but quite possibly nonspecific, initiates the disease. In the light of the findings reported in this paper, and the explanation offered for the occurrence of positive skin tests to pigment without sympathetic ophthalmia—namely, that after hypersensitivity to pigment is established there is no further absorption of pigment and hence no local hypersensitive reaction with the characteristic histologic picture—there are three obviously different ways of viewing the possible relation of pigment hypersensitivity to the pathogenesis of sympathetic ophthalmia.

These are (1) that once local ocular hypersensitivity to pigment has developed, the further or continued absorption of pigment produces a local hypersensitive reaction, clinically and histologically manifested as sympathetic ophthalmia. Thus some factor which favored or produced the continual absorption of pigment would be necessary for the outbreak of the disease. Such a factor might be related to the nature of the trauma, to some local condition, or to some specific or nonspecific infection.

The second explanation is that (2) after pigment hypersensitivity is established, some specific unknown infective agent initiates the disease. The further absorption of pigment is secondary and produces a hypersensitive reaction which determines the histologic picture of the disease, but is not

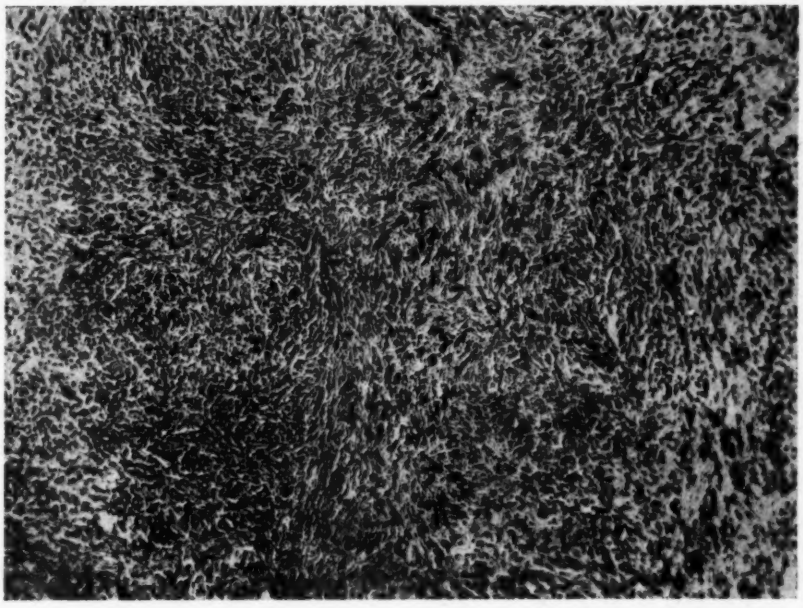

Fig. 6 (McPherson and Woods). Flat section of positive skin test.

per se the actual cause of sympathetic ophthalmia.

The third viewpoint would be (3) that the absorption of pigment after an ocular injury and the subsequent development of pigment hypersensitivity is a chance phenomenon and in no way related to the pathogenesis of sympathetic ophthalmia. However, when one considers the identical histologic picture of a flat section of the positive skin test and a flat section of the choroid in sympathetic ophthalmia (figs. 6 and 7), it does not seem

Fig. 7 (McPherson and Woods). Flat section of the choroid in a case of sympathetic ophthalmia.

probable that the last viewpoint is valid and that the pigment hypersensitivity is without significance. At the least, it would appear that it is the factor responsible for the characteristic and identical histologic picture.

Three of the five patients with Vogt-Koyanagi syndrome gave straight positive reactions and two patients gave negative reactions. It is interesting, although not statistically significant, that the incidence of straight positive reactions (60 percent) in this remarkable disease is almost exactly the same as the incidence of straight positives in sympathetic ophthalmia. The reason for the occurrence of positive reactions to pigment in patients with this disease must obviously be systemic absorption from the melanin-containing cells. That such absorption occurs in the eye is certainly not surprising. The disease is characterized not only by phagocytosis of the pigment of the uveal tract in the eye, but also by vitiligo and poliosis-bleaching and loss of pigment in the skin and in the hair. Here again, as in sympathetic ophthalmia, what precipitates the disease is not known. Both diseases may well be due to the same cause.

The doubtful or weakly positive tests present a problem. They occur with much the same frequency in sympathetic ophthalmia, postoperative and posttraumatic cases with and without a subsequent uveitis, and in uveitis from constitutional causes. When such reactions are encountered in sympathetic ophthalmia, they can easily be regarded as evidence either of a diminishing or developing pigment hypersensitivity, as the case may be. An examination of Table 1 shows that in sympathetic ophthalmia, although skin hypersensitivity may develop within the first month after the onset of ocular symptoms, in some cases it may not develop until the fourth month. Once developed, it may fade and disappear after a year. Thus there must obviously be both a developing and a waning phase, which in sympathetic disease would account for the weak or doubtful reactions. In other conditions in which doubtful positives occur—after injuries or operations with or without a complicating uveitis, and in uveitis from constitutional causes—one must hypothecate some different mechanism, either slight and insufficient absorption of pigment, or some unknown factors affecting either the antigenic activity of the pigment or the reaction capacity of the organism. In any event, the doubtful positive reaction in postoperative and posttraumatic cases and in uveitis from constitutional cause has no diagnostic or prognostic significance, occurring with the same general frequency in all three conditions.

Finally, what clinical significance should be attached to positive, doubtful positive, or negative skin reaction to uveal pigment? From this study, the answer seems fairly clear.

A positive test occurring after an injury or operation involving the uveal tract especially in the presence of a persisting traumatic uveitis should put the ophthalmic surgeon on guard. A positive reaction does not mean that sympathetic ophthalmia is imminent or inevitable. It does mean that it is a possibility. This possibility should be carefully weighed in the decision to enucleate or not to enucleate the injured eye. In general, our feeling is that if all is going well and it is probable that the injured eye will have some useful vision, then a positive pigment test should be disregarded. On the other hand, if the eye appears clinically as a possible sympathogenic eye, or if there is little prospect for the preservation of any useful vision, then enucleation is indicated.

The interpretation of a doubtful positive reaction is more difficult. In outspoken sympathetic ophthalmia, the doubtful or weak positive may be regarded as evidence either of a developing or waning hypersensitivity. In conditions other than frank or suspected sympathetic ophthalmia, the doubtful positive reactions have obviously little diagnostic significance.

Negative or nonspecific reactions give

only slight comfort to the surgeon. A study of Table 1 reveals that in four of the 30 cases of sympathetic ophthalmia tested for skin hypersensitivity in the first three months, the skin test was negative when first done, although in two patients it later became positive. In these patients, the development of hypersensitivity in the skin apparently lagged behind its development in the eye. Thus a negative test after an injury does not preclude the possibility of sympathetic ophthalmia. However, in the presence of a reasonably favorable clinical course, a negative test does make the occurrence of sympathetic ophthalmia very unlikely.

Summing this up, the diagnostic and prognostic significance of the skin sensitivity test is limited. Positive tests are causes for alarm and increased vigilance. Doubtful and negative tests lend slight comfort and indicate in general a better prognosis. However, the margin of error in both positive and negative tests is so great that their interpretation should be made only in the consideration of the clinical course.

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FUNCTIONAL HOME EXERCISES IN CASES OF EYESTRAIN

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The criterion for giving or not giving orthoptic treatment does not depend upon the presence of a demonstrable heterophoria, but upon the presence of an actual asthenopia. If the patient complains of symptoms after refractive correction, orthoptic treatments are indicated, but if there are no symptoms in spite of the presence of heterophoria, such heterophoria being of the inherent type, then treatments are contraindicated. Inherent exophoria may be due to anatomic causes, and the etiology of this condition indicates that, normally, it does not have a related asthenopia.

A developed exophoria of more recent onset shows, on the other hand, that a breakdown has occurred in the functional ability of the eyes to restrain a latent deviation by means of such reflexes as fusion, convergence, and so forth. It is the persistent effort of the subconscious mind to maintain normal binocular vision in the presence of ocular imbalance which causes the development of symptoms. These symptoms may range from slight discomfort to extreme neurogenic fatigue and the production of psychoneuroses. Treatment is therefore directed toward the attainment of a smoothly working dynamic apparatus, so that the individual can deal both physiologically and psychologically with any visual situation in any presentation and still be within the limits of ocular comfort.

In all cases there must be a full range of convergence, but in exophoria and esophoria the latent deviation must also be dealt with by actually varying the accommodation-convergence relationship for any given distance. In exophoria, besides teaching full convergence over the normal range, we endeavor to train excess convergence for a given amount of associated

accommodation; or, in other words, to teach relative relaxation of accommodation while the convergence is exerted to the utmost.

In the case of esophoria, it is still necessary to teach full convergence, since this is a normal function, together with a lessening of the rigid accommodation-convergence link which exists. In this way the esophoric patient is taught to accommodate without overconverging or while relaxing the associated convergence. A full range of divergence must also be present. In both exophoria and esophoria, the patient acquires a foveal suppression habit, especially in the case of latent divergence. Antisuppression treatment is, therefore, always necessary.

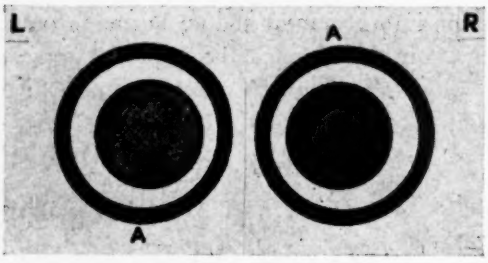

Fig. 1 (Eyles). These figures are printed on separate white cards which are 2 by 3 inches in size. The center circle of the card marked L is colored a medium green; that of the card marked R is a medium red.

It is noted that when the normal ocular functions such as convergence, divergence, good fusion range, and stereoscopic vision are fully restored, there is complete freedom from symptoms even though a heterophoria may still be demonstrated by disassociation tests.

Severe symptoms may be present even when there is orthophoria for distance and near, in which case convergence insufficiency is usually the cause. The convergence reflex was the last to develop phylogenetically, and it is often easily disrupted either under the strain of excessive use or through under use, as in the case of the faulty ocular rigidity (almost a "fixus") which some persons acquire. These persons apparently have a horror of ocular movement, especially disjunctive. The treatment of convergence insufficiency is very satisfying, since symptoms quickly disappear.

Another group of patients, the presbyopic, need some functional adjustment. Their reading addition is conducive to the increase of exophoria and, since accommodation is not normally used, to the lack of convergence incentive.

Orthoptic treatments should prove much more effective than the use of base-in prisms, in correcting these various conditions. Functional anomalies require a dynamic functional cure rather than a physical adjustment which cripples the normal innervational and physiologic processes. Because of this, orthoptic treatments attempt to establish corrective conditioned reflexes, or to reinforce existing conditioned reflexes, on a sound physiologic basis.

Exercises for home use

The following exercises (to be carried out as the doctor recommends) are intended primarily for home use. In them an attempt has been made to formulate a graded series for the elimination of suppression and the development of full convergence, full divergence, controlled convergence-accommodation relationships and stereoscopic appreciation. The patient is given the six cards* (figs. 1 to 3), together with a page of directions. The technical notes which follow the exercises are a useful supplement for the ophthalmologist or orthoptic technician.

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*Dr. Walter B. Lancaster has helpfully suggested that each pair of pictures may be attached to a piece of cardboard and, when the patient has learned to fuse them, the cardboard can be cut in two and the pictures held one in each hand and then separated laterally.

Two minutes only are to be spent on these exercises at a time. Do them twice daily until you can do them all, then once daily for one week, followed by once every other day for a week.

Exercise 1. Hold the first pair (fig. 1) of cards side by side in your hand at about three quarters of an arm's length, or reading distance. Hold a pencil with the other hand, just in front of the cards and in the center of them. Look at the pencil point as you gradually move it toward your eyes. You will notice four blurred sets of circles in the background instead of two. When the pencil is about halfway between the pictures and your eyes, the two middle sets of circles will join up, making three in all. Hold the pencil still, and notice that the middle picture is a tunnel and has "depth"; that is, you are looking into it. There is an "A" above and below the tunnel. Keep looking at this tunnel and you will be able to remove the pencil without losing the picture. Is the middle of the tunnel red or green, or a mixture of both colors? Make a conscious effort to see both red and green.

Exercise 2. Watch the tunnel obtained as in Exercise 1, and bring the cards toward your eyes, almost touching your nose, then back to arm's length. Try to see the tunnel clearly.

Exercise 3. Watch the tunnel at about three quarters of an arm's length, and part the cards sideways, slowly. The tunnel will become smaller and smaller until the pictures break into four.

Exercise 4. Hold the cards close to your eyes, touching your nose, and you will see a blurred tunnel with its center red and green. Withdraw the cards from the eyes, slowly, watching the tunnel which will become clearer. A red and green picture will appear on either side of the joined picture.

Exercise 5. If you find Exercise 4 difficult, do this one. Hold the cards at three quarters of an arm's length or reading distance and look beyond them. You will notice three sets of circles. Watch the middle tunnel, which is the joined picture.

Exercise 6. In both Exercises 4 and 5, the tunnel, which is the middle joined picture, appears to be inverted; that is, the small circle is nearer to you than the larger circle. The center of the circle should be both red and green fused. See it clearly, and bring the cards toward your eyes and back again to arm's length.

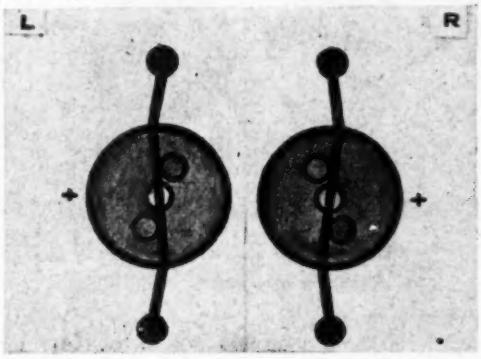

Fig. 2 (Eyles). The card size and figure colors are as described in Figure 1. The three tiny circles are colored as follows: top circle, purple; center circle, white; bottom circle, orange.

Exercise 7. Still watch the tunnel, and part the cards sideways. It is possible to part them about one inch before they break into four.

Exercise 8. Take cards Number 2 (fig. 2). Repeat Exercises 1 to 3. In this case notice that in the middle picture the line from the top ball comes toward you, then turns backward through the circle, and finally returns toward you and the lower ball. There is a small cross on either side of the middle picture.

Exercise 9. Obtain the middle picture as before, and look from the purple ball to the yellow ball and vice versa six times. The yellow ball is nearer to you.

Exercise 10. Do Exercises 4 to 7 with

cards Number 2. The stereopsis is reversed.

Exercise 11. Obtain the middle tunnel as in Exercises 4 and 5. Look from the purple ball to the yellow ball and vice

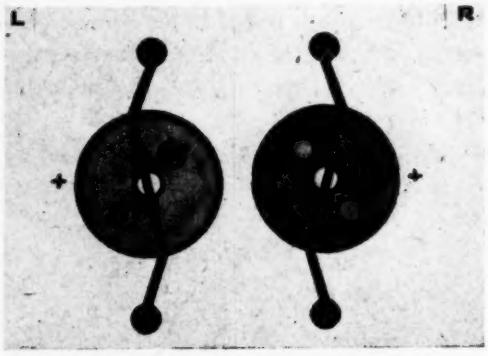

Fig. 3 (Eyles). The size and all colors are as indicated in Figures 1 and 2.

versa. Now the purple ball is nearer.

Exercise 12. Repeat all with cards Number 3 (fig. 3).

Technical notes

Exercise 1. For training convergence and fusion. Antisuppression exercise. If the middle picture appears green, then there is suppression of the left eye, and the right eye is the master eye. If the middle picture appears red, then there is suppression of the right eye, and the left eye is the master eye. If it varies, then there is alternating suppression. When there is suppression of one eye, the patient should concentrate on seeing the suppressed color. This is a good exercise for suppression on convergence.

Exercises 2 and 3. For training convergence, and relaxing accommodation while converging. Especially for treating exophoria and accommodational spasm at near (ciliary spasm), and presbyopia with convergence insufficiency.

Exercises 4, 5, and 6. If the middle picture appears green, there is suppression of the right eye. If the middle picture appears red, there is suppression of the left eye, in divergence.

Exercises 4 to 7. Especially for treating esophoria. Produces accommodation while relaxing convergence. Exercises divergence.

Cards Number 2 and 3. Angles of fusion of the two lines are 5 and 10 degrees. This is a difficult fusion exercise. Looking from the purple to the yellow balls is good convergence exercise while maintaining the static convergent position in Exercise 9 and the divergent position in Exercise 11. In all exercises depth perception is exercised.

Exophoria. Exercises 1, 2, and 3 are necessary to obtain good convergence. Relax the eyes by finishing with Exercise 5.

Esophoria and ciliary spasm. Exercises 1, 2, and 3 to obtain smooth convergence. Concentration upon Exercises 5, 6, and 7 to lessen th esophoria, ceasing Exercises 1, 2, and 3 as soon as possible.

Convergence insufficiency and presbyopic adjustment. Exercises 1, 2, and 3 especially. Relaxing with Exercises 5, 6, and 7 afterward.

Hyperphoria. Fusion of the two cards is obtained by raising one of them a little. Vertical vergences are exercised by gradually lowering the card previously raised, and keeping the tunnel fused.

Conclusion

A fusional reserve well beyond ordinary requirements will be built up by these exercises.

Since the muscle balance often changes quickly after doing them, the ophthalmologist may find it advisable to check the patient's measurements at least once every two weeks.

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THE VERGENCE TEST—AN EVALUATION OF THE VARIOUS TECHNIQUES*

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The vergence test constitutes an important phase of a routine refraction. It offers very tangible evidence as to the binocular efficiency.

In spite of the importance of the vergence test in routine refraction, there seems to be a question as to how generally the test is used. There apparently exists, in the minds of many, a question as to the reliability of the test. Moreover, they feel that the information obtained does not justify the expenditure of time. This attitude is seemingly due, in a large degree to the variability in results obtained. Such a reaction is understandable, because we know that a wide variation in readings may exist, not only between the different patients tested, but even in retesting the same patient. Undoubtedly, the test would be used more routinely if the reading would check on repetition; if different patients would respond similarly; and if examiners could depend upon each other's findings. In other words, definite, dependable standards should be established.

UNIFORMITY OF PROCEDURE NEEDED

Granting that various factors enter into the problem, it is evident that one factor which definitely leads to variability in results is the lack of uniformity of procedure. Analysis of available data shows a wide variation in both technique and interpretation of the findings. Such a situation inevitably leads to confusion.

An efficient technique should be evolved which is standardized. In addition, the clinician should have a clear conception as to the interpretation of the results and their relative value in contributing to the analysis of the case being examined. With this standardization of technique, and the resulting greater consistent readings, the clinician would be more willing to spend precious time to do the test.

It is the object of this presentation to attempt to analyze the more common methods of testing vergence in use today, and to attempt to evaluate them. It seems logical that a definite technique could be evolved which would embody all the advantages of the various techniques, and be a step toward securing more constant and dependable results.

The problem may be divided into two phases: first, a consideration of the various steps to be determined in the vergence test; and, second, the technique for determining these steps.

VARIOUS STEPS TO BE DETERMINED

In the first consideration, which has to do with the various steps to be determined, there seems to exist a difference of opinion as to what constitutes a satisfactory procedure. A review of the medical literature shows that very little organized investigation has been carried out on the subject. Judging from the information available, a large percentage of the profession employs the vergence test, but varies its procedure. For example, Pascal[1] and Roper[2] advocate that the blur, break, and recovery points should be determined; whereas, Tait[3] and Burian[4] advocate the determination of only the break and recovery points. As a whole, it appears that most clinicians limit their observation to only the break point.

*Read at the 82nd annual meeting of the American Ophthalmological Society, San Francisco, California, June, 1946. This paper was accompanied by elaborate charts, information concerning which may be obtained from the author.

The majority of the literature available is comprised of the work of physiologists, and this work was instigated chiefly by Sheard[5] and Fry.[6] Their research, which is based upon investigation both in the laboratory and in the clinic, is as a whole in agreement. According to their data, there is evidence to support the fact that an adequate vergence test should include a test for adduction, which embraces the determination of the blur, break, and recovery points for both distance and near. In testing for abduction, the determination of the blur, break, and recovery is made for near, but only the break and recovery for distance. In the case of the supraduction, only the break and recovery are determined. This group of observers maintains that a complete picture of the vergence power is obtainable only when the above routine is followed.

Physiology of the vergence test

Although it is outside the scope of this paper to explain the physiology, and discuss the various interpretations advanced concerning these various steps, it seems advisable to summarize very briefly the consensus held by the majority of the workers in this field. It is thought that the chief contribution of the blur, break, and recovery points give to the analysis of the case is a more detailed picture of the accommodative-convergent relationship. In them, we have a means of analyzing the various phases of this association, bringing to light any abnormal relationships.

Fry[7] states, "In the blur-point test, prism power is gradually increased in an effort to throw the images off corresponding points of the two retinas. The reflex fusional mechanism compensates the effect of the prisms by making the eyes turn as the prism power is increased. As long as the increase in the power of the prism is compensated solely by the operation of the reflex fusional mechanism, the target remains clear. When blurring does occur, it is the limit of fusional convergence, and means that the accommodative mechanism is thrown out of focus by over-accommodation in the base-out test, and under-accommodation in the base-in test. These changes in accommodation assist in maintaining fusion, because every time accommodation increases or decreases, the associated convergence must follow suit.

"Accommodation remains relatively constant from the base-in to the base-out blur point. At the base-out blur point a rapid increase in accommodation occurs, and at the base-in point a rapid decrease. These changes in accommodation continue at the same rate all the way from the blur point to the break point. Positive fusional convergence is used to bring the eyes from the phoria position to the base-out to blur position and from this point on an increase in convergence is associated with it. Negative fusional convergence is required to bring the eyes from the phoria position to the base-in to blur position, and from this point on accommodation must be decreased in order to obtain further divergence of the eyes."

The break point represents the maximum convergence or divergence effort which can be made in order to maintain single vision. It represents the extent of the reserves set aside by the brain to hold the function steadily in force. Addition of prism power from blur to diplopia gives an additional measure of the degree to which accommodation and convergence are associated.

The recovery, or the reversion to fusion, involves the sensitivity of retinal elements in the extra-macular region. It estimates the efficiency and rapidity with which binocular fixation adjustments are made, and it probably has no bearing on the efficiency with which single binocular vision is maintained after being once established for a given fixation. It gives a measure of the "desire" for binocularity.

If the patient takes the attitude of doing all he can to recover fusion, the recovery finding will be much higher than if he had taken a passive attitude and waited for the two images to fuse of their own accord. When such an effort is made to fuse the images, presumably what happens is that a voluntary movement of convergence occurs first, and then as soon as the diplopia is reduced to the point at which the reflex fusional mechanism can operate, this mechanism helps to complete the recovery of fusion.

Fry[8] states, "The movement to recovery fusion is often referred to as a reflex. The application of like stimuli to non-corresponding points of the two retinas is supposed to constitute the adequate stimulus for evoking the reflex. The nearer the two stimuli come to falling on corresponding points, the more effective they are in evoking the reflex, and if the image on one retina falls too far away from the point corresponding to the image on the opposite retina, no response can be obtained. Undoubtedly, the operation of such a reflex mechanism must play a dominant role in the reversion to fusion, but it is necessary to reckon with a certain amount of voluntary control over convergence."

Interpretation of abnormal findings

This very briefly indicates some of the opinions held concerning the physiology of the vergence tests. To elucidate on the interpretation of the various abnormal findings would exceed the scope of this paper. It is noteworthy that experimental data reported attribute practical significance to the various abnormal relationships.

The absence of a normal accommodative-convergence relationship indicated by a high, low, or absence of the blur point, is of diagnostic significance. Likewise, a high or low break point and a high or low recovery point is of value in estimating the binocular efficiency.

Fully cognizant of the many valuable contributions to the subject, it is obvious that the final status of the blur, break, and recovery points has not been fully established. As long as there exists a disparity of opinion concerning their value, the subject is still unsettled and calls for more study.

Fry,[9] who is one of the outstanding contributors, states, "I am very humble about this problem, and if I heeded my scientific inclinations, I would not try to formulate an opinion. But from the point of view of teaching students, it is necessary to formulate some statement of the problem, and the formulation that I have presented is really one which we are accepting tentatively as a basis of clinical procedure until it can be replaced by a more satisfactory formulation of a problem."

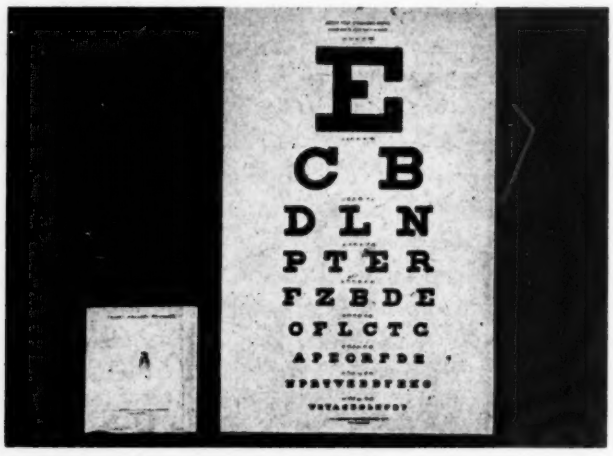
A unified effort should be made to establish a definite evaluation of the various phases of the vergence test. If the results are based upon an adequate scientific background, the status of the test will become fully established, and we will know what is of practical value and what is of scientific interest only. A common understanding will be reached in deciding as to what routine should be followed in order to obtain the maximum practical knowledge in doing the vergence test.

Methods of doing vergence test

The other consideration, namely, the method of doing the vergence test, is an important factor in our problem. The literature shows a wide variation in techniques and instrumentation. For example, the use of the prism is typical. Some advocate a rotary prism, others advocate the prism bar; whereas, others advocate a separation of the target used. The most commonly used target is a spot of light; others use various types of targets, such as a single letter, a line of letters, or a complete chart such as the Snellen chart, all of which are constructed to stimulate

macular fusion with more or less disregard for perimacular and peripheral fusion.

It is evident that such variation in method should produce variable readings, and thus make it impossible for two examiners to compare results. It is logical to believe that the various techniques are not equally accurate. Steps, therefore, should be taken to select the most efficient method and to make it a standard. If a standardized technique were adopted, more uniform results could be expected. This, in turn with a knowledge of the interpretation of the findings, would stimulate the examiner to make the vergence test a part of his routine investigation, and consider it as important as testing for astigmatism.



A B

Fig. 1 (Fink). A is a reduced Snellen chart to be used at near point. B is a standard Snellen chart.

Standardization study undertaken

With this in mind, the following investigation was undertaken. One hundred cases were analyzed and the data obtained were used in conjunction with other available data to formulate a method of procedure. Various techniques and instruments were compared, both for the type of prism used, and also the type of target most suited. It was considered desirable to eliminate all factors in instrumentation which would contribute to variation in readings.

Refractive procedure followed

All cases were refracted under cycloplegia, and only the cases with 20/20 vision in each eye and a small refractive error were selected. In addition, only cases showing a small amount of heterophoria, normal accommodation, and PcB. were used. By being selective, a greater possibility of what may be considered normal vergence range could be obtained.

The procedure was as follows: The full refractive error was worn during the examination. Accommodation for each eye was tested. Near point of convergence was likewise checked. Muscle balance was determined by the prism-displacement method. This was checked for both distance and near. Only cases whose muscle balance were within what may be considered normal limits were employed. Cases showing a hyperphoria greater than one prism diopter were discarded. The cases showing one diopter of hyperphoria had it corrected by prisms.

VERGENCE-TEST PROCEDURE

In doing the vergence, the jump method (prism bar) was compared with the results using the rotary prism. Adduction and abduction were measured both for distance and near.

Only the lateral ductions were included in the investigation, because the additional testing of the vertical ductions would prolong the test, producing more fatigue, and would not appreciably influence the conclusions.

In checking the vergence at 20 feet, the following steps were taken: For adduction—the blur, break, and recovery points were determined. For abduction—the break and recovery points were determined. At 15 inches both for adduction and abduction the blur, break, and recovery points were recorded.

The following targets were compared both for distance and near. A standard spot of light was employed both at 20 feet and at 15 inches. A standard Snellen chart was used for the 20-foot determination, whereas, the reduced Snellen chart was used at 15 inches.

A third target, as devised by the author, was used for comparison. This consisted of a line of letters corresponding to the 20/20 line such as used on the Snellen chart. Behind this line of letters was a large "E" one foot in diameter, which was rotated slowly by a small motor. A similar chart was used for the 15-inch test in which small print was used, with a rotating "E" four inches in diameter placed behind it.

The fourth target compared was one in which stereoscopic vision was employed. Polaroid lenses were placed in the phorometer, and the patient was tested at 20 feet by projecting a picture on a screen. This process was repeated at 15 inches. When the patient was tested at 20 feet, the room was in a darkened condition so that objects in the room would not influence results. It is the opinion of some students of this subject that by using stereoscopic vision we simulate ocular experience encountered in daily life where the whole retina is involved. They maintain that binocular action is dependent not only on the macula, but also on perimacula and peripheral areas of the retina, and a true test of the vergence power should be the result of such a stimulation of the entire retina, rather than merely macular.

A B

Fig. 2 (Fink). A is a rotating target to be used at near. B is a rotating target to be used at distance.

The cases examined had preliminary training so that they had a clear understanding of what was expected of them. The element of fatigue was carefully avoided. Some cases were discarded when the coöperation was questionable.

Discussion

An analysis of the data obtained from the study suggests the following conclusions:

It is important to emphasize that fatigue will alter the findings. The test should therefore be carried out with a minimum number of repetitions. When fatigue is evident, rest should be taken.

Proper rotation of the prisms should be employed. Rotating the prism too rapidly is apt to break the fusion quickly, resulting in abnormal duction findings. Likewise, too slow a rotation places undue strain on the muscles involved, and will also alter the findings.

The full correction should be employed, including the correction of the vertical imbalance of one degree or over.

Rotary prisms seem to be superior to the prism bar or separate prism. The gradual increase in using the rotary prism gives a more constant result than the steplike increase as experienced when using the prism bar. The phorometer is the most satisfactory instrument for this.

The test should be done at both 20 feet and 15 inches. The 15-inch test is the more informative.

To obtain the maximum information, it is necessary to test the blur, break, and recovery points. In distance testing with the base-out prism, the three steps should be noted. With the base-in prisms, only the break and recovery points are noted. In testing at 15 inches, the blur, break, and recovery points are noted for both base-out and base-in tests.

In testing for the blur points, great care must be exercised. Considerable difficulty is encountered frequently in the recognition of the blur point, especially when the blur and break occur almost simultaneously. The patient should concentrate on the smallest print he can see, and indicate the point when the print is completely blurred. By using this technique, a more definite reading can be obtained. The blur point can be simulated by inserting a half-diopter sphere before the eyes, which will cause the letters to blur. This will give him a better conception of what to watch for.

The break point is comparatively easy to determine, and is more consistent if the attention is directed to the entire target when the blur point is reached. The patient is instructed to hold the target single as long as possible. When the break occurs, he will be suddenly aware of two widely separated targets and may or may not have been conscious of the process of separation of the images.

The recovery point is variable if a definite technique is not carried out. The patient is instructed to concentrate on the whole target and to try to unite the separated images, thus having convergence take an active part. This convergence, plus the fusion desire, causes the establishment of single vision.

The target producing the most consistent results was the rotating type. It was superior to the others in that the motion produced a more consistent stimulus to the perimacular area, so that when the break point was reached, the patient was immediately conscious of it. Likewise, the recovery-point reading was more consistent because the stimulus was uniform. It should be emphasized that the recovery-point reading is very uncertain and variable unless the patient is fixing on the entire target, which will stimulate both the macula and perimacula. To fix on small letters at this point materially alters the reading.

Next to the rotating target in efficiency was the Snellen chart. The regulation chart was used for distance and the reduced chart for close. From a practical standpoint, this target can be used, although the results were not so consistent as the rotating target. However, it was superior to the spot of light.

The spot of light was the most inefficient. It produced a wide variation in results because it stimulated only the macula, ignoring the perimacular area; reflexes confused the patient; the bright light fatigued the patient; the blur point was practically impossible to determine; the break and recovery points were frequently missed and varied widely. Repeated trials were frequently necessary which added to ocular fatigue.

The stereoscopic target using polaroid seemed to offer even greater possibilities than the other targets. It has the advantage in that it tests the macular area, the perimacular, and also the peripheral area of the retina. It thus simulates what we actually experience in daily life. It gives us a truer picture of our ocular coördination than the more artificial methods. It is possible that with this method the rotary prism should not be used, but rather the vergence power tested by a separation of the two superimposed pictures making up the target. This method, however, is as yet not feasible, but it is very possible that it will ultimately be the method of choice. This change in technique should produce more consistent results, and will give us a truer picture of the efficiency of the binocular apparatus.

The comparison of the polaroid method with the rotary target was favorable. The readings did not agree in amount but the relationship in readings was in fair agreement. The objective is quickly grasped by the patient, and the blur, break, and recovery points recognized with comparative ease. Of the various targets tested, it seems to offer the greatest promise because it simulates more closely conditions in daily life, and is a better stimulus to our desire for stereoscopic vision.

Summary

To formulate a routine to be followed in testing the vergence, the following procedure is suggested. Based on the study of the above cases, it incorporates techniques which seem to produce more consistent results, are more understandable to the average patient, lessen the time factor, produce less fatigue, and give the maximum information obtainable.

1. The test is briefly explained to the patient.
2. Fatigue is avoided. A short interval of rest is valuable if the patient shows signs of fatigue.
3. The room should be quiet, and the period free of interruption.
4. The room is illuminated in a normal manner.
5. The correction is used, and if the hyperphoria is one diopter or over, it should be corrected.
6. The phorometer with rotary prism is preferable.
7. The prism should be rotated at the proper speed.
8. The rotating target or the Snellen chart is used both for distance and near.
9. Distance base-in test: Attention is directed to the whole chart and not to a specific line. Base-in prisms are rotated before the eyes in the manner indicated above. A recording is made of the amount of prism necessary to create diplopia. The rotation of the prisms is then reversed, having the patient make an effort to unite the images and the point recorded as the return fusion occurs.
10. Distance base-out test: A similar method of procedure is used in making this test as was used in the base-in test, with the exception that we employ a line

of small letters representing the patient's highest distance visual ability. The selection of the smallest type the patient can read is of extreme importance, because even a slightly larger fixation object is apt to give a much higher reading.

The patient's attention is directed to this line of small letters; base-out prisms are introduced in increasing amounts; and the point recorded at which the patient reports he can no longer read the letters. This is the distance blur-out point.

Next, direct the patient's attention away from the small blurred letters to the entire target and instruct him to keep it single as long as possible. The base-out prisms are increased in amount and the point recorded at which diplopia occurs. The patient is asked to try to unite the two targets, and the prisms are then rotated in a reverse direction. A record is made of the amount of prism, allowing for refusion of the target.

11. Near-point base-out test: The technique is similar to that used in testing the distance base-out test. The patient's attention is directed to the reading of the smallest type possible on the near-point target. Base-out prisms are slowly increased in amount before the eyes as the patient attempts to maintain the necessary accommodative adjustments for keeping this fine type legible. The point at which the type becomes illegible, or blurs out, is the blur-out point.

After the blur-out point has been reached, the patient's attention is referred to the entire target, and he is instructed to keep it single as long as possible. The increase of base-out prisms is continued until diplopia results. This is the break point. The prismatic power is next reduced and the patient is instructed to fuse the two images. This point is recorded as the recovery point.

12. Near-point base-in test: The technique for this test is identical with that of the foregoing test, with the exception that base-in prisms are used.

13. Vertical ductions: These are tested for the break and recovery points both for distance and near.

This briefly outlines a feasible routine to be used in testing the vergence. Appreciating the fact that such a procedure is time-consuming, and that such an expenditure of time may not be indicated in every refractive case, it will, when the indication arises, serve as a guide to a complete analysis of the vergence power, and give a more complete picture of the accommodation-convergence relationship. Although the above recommendations are based on a comparatively small group of cases, and therefore cannot be considered except as suggestive, they serve at least to point the way to further investigation. It is important to point out that the inclusion of the blur, break, and recovery points in the above routine is tentative. The data obtained concerning them in the series of cases here presented are suggestive. More data are necessary before a final opinion can be formulated concerning their clinical value.

Conclusions

The vergence test is an important part of our daily routine. It is generally admitted that the various techniques used for this test do not give consistent results. An attempt is made to determine which is the most efficient and the most dependable. Certain changes in techniques seem to increase the efficiency of the test. These changes in techniques, according to the data, produce more dependable readings, produce less fatigue of the patient, are more understandable to the patient, save time for the examiner, and give a more correct interpretation of the findings.

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INTRAOCULAR FOREIGN BODIES IN SOLDIERS*

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Retained foreign bodies were found in 731 of 3,882 eyes of soldiers studied at the Army Institute of Pathology during World War II. Although nearly all of these foreign bodies were introduced during combat and training, a few were the result of preinduction injuries, with recent exacerbations of pain and inflammation leading to enucleation. All globes with penetrating wounds were examined by X ray and were searched for foreign bodies. In many instances the particles were so small or so deeply embedded in organizing hemorrhage or inflammatory membrane that they were not recovered from the gross specimens and only became visible on microscopic examination. These could not be subjected to magnet test, but sections containing them were stained with Prussian blue. Those which gave a positive iron reaction were classified as probably magnetic, although the degree of their magnetic quality could not be determined; whereas, those giving no reaction were regarded as nonmagnetic. The Federal Bureau of Investigation assisted in the more specific identification of many of the foreign bodies by spectroanalysis. In many eyes the particles were multiple (figs. 1 and 2), and in a few, both magnetic and nonmagnetic materials were found. The following table demonstrates a preponderance of retained nonmagnetic foreign bodies in eyes of soldiers who, in all but a few instances, had sustained military injuries.

INTRAOCULAR FOREIGN BODIES

Ferrous	273
Nonmagnetic	452
Both	6

Ferrous foreign bodies (figs. 2 and 3), although outnumbered by nonmagnetic ones, formed the largest relatively homogenous group. Some were more strongly magnetic than others, and it is possible that many which were weakly magnetic on direct contact may have been clinically nonmagnetic.[2] Siderosis, with deposits in the cornea (fig. 4), lens (fig. 5), filtration angle, iris, ciliary

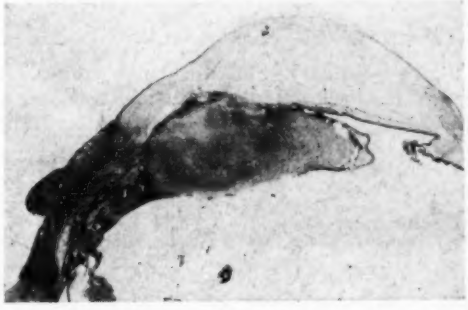

Fig. 1 (Wilder). Multiple ferrous foreign bodies in cornea, ciliary region, lens and vitreous. Scar of entrance of intraocular foreign bodies at the limbus. Enucleation 23 days after injury in action by bomb explosion. A.I.P. Negative 97290.

* From the Army Institute of Pathology.

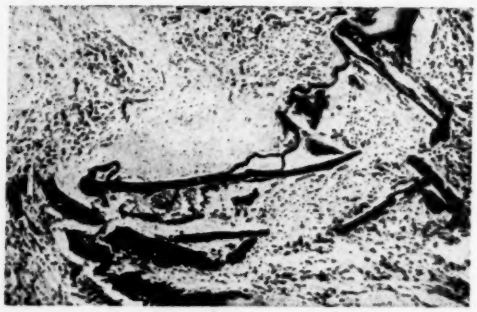

Fig. 2 (Wilder). Multiple ferrous foreign bodies in an inflammatory cyclitic membrane. Magnet extraction of one steel particle was performed on the day of injury six weeks before enucleation. A.I.P. Negative 99319.

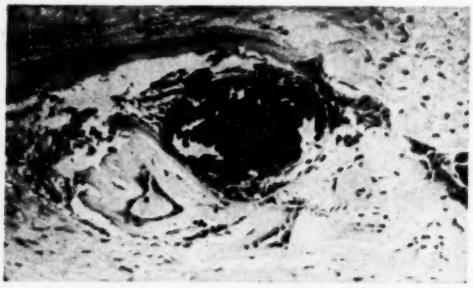

Fig. 3 (Wilder). Ferrous foreign body in lens capsule incarcerated in an inflammatory cyclitic membrane. Enucleation one year following a penetrating wound by a screw driver. A.I.P. Negative 99322.

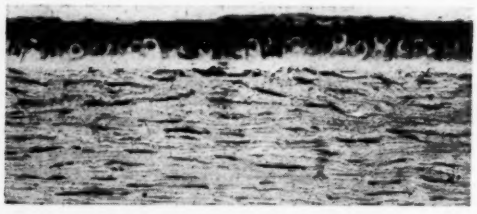

Fig. 4 (Wilder). Siderosis bulbi, cornea. Iron pigment phagocytized by the cells of the interlamellar spaces. Enucleation two years after injury. A.I.P. Negative 100247.

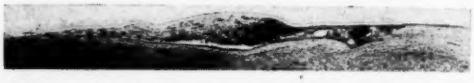

Fig. 5 (Wilder). Siderosis bulbi, lens. Iron pigment in an anterior capsular cataract. Enucleation nine months after injury. A.I.P. Negative 100248.

epithelium (fig. 6), and retina (figs. 7 and 8), occurred in 27 eyes with retained ferrous particles. In one eye, a rather extensive iron reaction was noted only 23 days after injury, but in this case many ferrous particles provided multiple foci of diffusion. In some instances in which ferrous foreign bodies had been retained for much longer periods, early encapsulation by scar tissue (fig. 9) had prevented dissemination of the metal, and only a local iron reaction was obtained with Prussian blue. In hematoxylin and eosin sections basophilic staining, as well as pigmentation, was frequently ob-

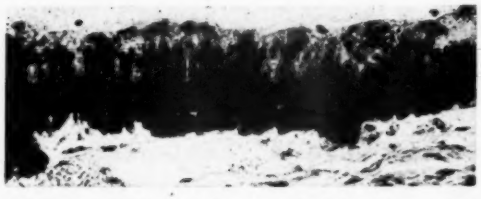

Fig. 6 (Wilder). Siderosis bulbi, ciliary epithelium. Iron pigment in the normally nonpigmented inner layer. Enucleation 11 months after injury. A.I.P. Negative 100246.

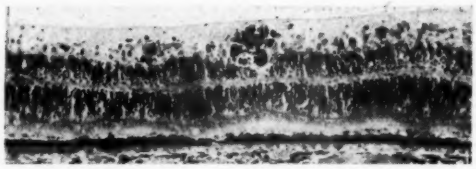

Fig. 7 (Wilder). Siderosis bulbi, retina near equator. Iron pigment around a retinal vessel. Enucleation two years after injury. A.I.P. Negative 99252.

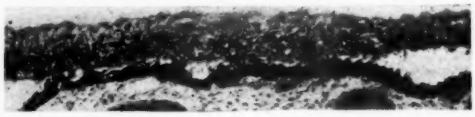

Fig. 8 (Wilder). Siderosis bulbi, peripheral retina. Iron pigmentation and atrophy. Enucleation two years after injury. A.I.P. Negative 99250.

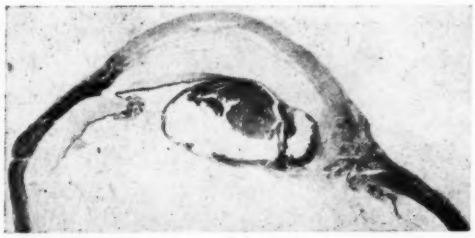

Fig. 9 (Wilder). Scar tissue around the site of a ferrous foreign body in the posterior chamber preventing the dissemination of iron pigment. Enucleation two months after injury. A.I.P. Negative 97284.

served around the sites of the ferrous material (fig. 10), but rarely around other metals and never to so marked a degree.

The nonmagnetic foreign bodies comprised a variety of materials. Among the metal substances, copper, brass, lead, nickel, and zinc alloy were identified; among the nonmetallic, glass, vegetable matter, eyelashes, rock, shale, and clay predominated.

Copper and brass were most numerous among the nonmagnetic metals. Unfortunately chalcosis could not be demonstrated by special staining, and it was, therefore, impossible to study the intraocular distribution of copper.

Many nonmagnetic foreign bodies were readily identified by gross or microscopic examination more specifically than by magnet test. Copper fragments (fig. 11) could be recognized both in microscopic sections and on gross examination by their characteristic color. Lead (fig. 12), in comparatively large pieces, was sectioned in the celloidin blocks with little damage to the microtome knife. Glass, of course, was easily identified on gross examination, and vegetable matter and eyelashes (fig. 13) on microscopic examination. Nonmagnetic foreign bodies were found not only as primary missiles or in association with magnetic material, but also as material retained after extraction of a magnetic missile or as implants along the path of a primary missile which had passed completely through the eye. Eyelashes were the most common of the secondary foreign bodies. In one instance a fragment of chrome-green paint remained in the choroid, although the piece of metal from the bumper of a jeep which it had camouflaged had been extracted by magnet. Intraocular particles of stone, clay, and vegetable matter frequently were retained after land-mine explosions. In severe head injuries, skin (fig. 14) and bone (fig. 15) were sometimes im-

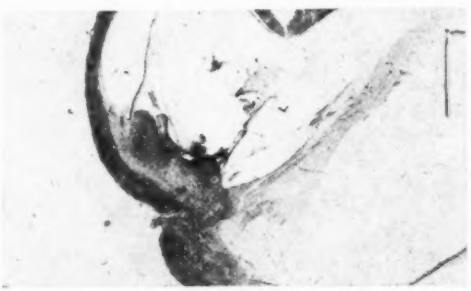

Fig. 10 (Wilder). Iron pigment and basophilic staining around the site of a ferrous foreign body in the vitreous chamber. Enucleation 41 days after injury. A.I.P. Negative 97286.

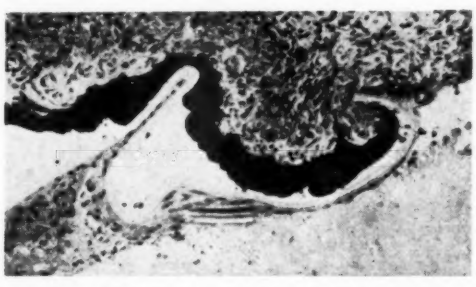

Fig. 11 (Wilder). Copper wire in early anterior capsular cataract. Lens capsule recoiled outward at site of entrance. Chronic iridocyclitis with posterior synechia. Enucleation six days after injury. A.I.P. Negative 99321.

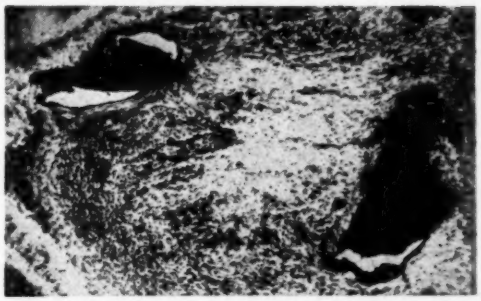

Fig. 12 (Wilder). Lead in inflammatory tissue in the vitreous chamber. Enucleation five months after injury. A.I.P. Negative 99318.

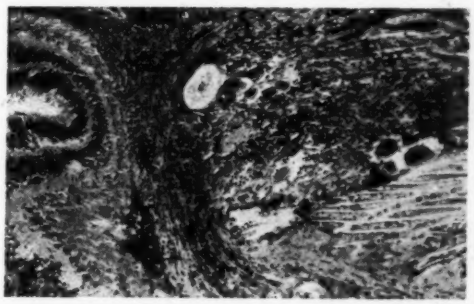

Fig. 13 (Wilder). Vegetable matter and cilium in inflammatory tissue in the vitreous chamber, following a land mine explosion. Enucleation two months after injury. A.I.P. Negative 97289.

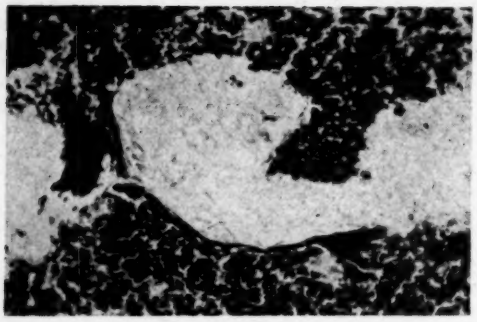

Fig. 14 (Wilder). Epidermal epithelium with keratic debris in a vitreous abscess. A zinc alloy primary missile and hairs were also retained until enucleation four days after injury. A.I.P. Negative 97361.

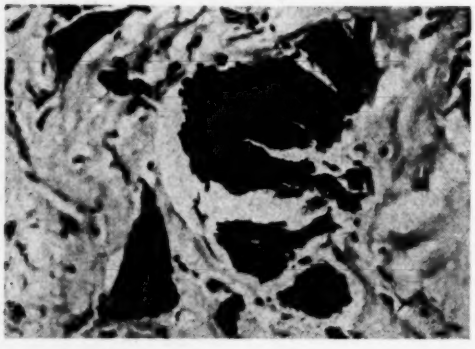

Fig. 15 (Wilder). Implanted bone incarcerated in a cyclitic membrane following a skull wound. Enucleation nine months after injury. A.I.P. Negative 97356.

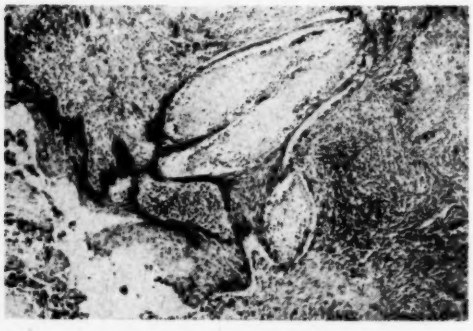

Fig. 16 (Wilder). Hyperplastic conjunctival epithelium around a thorn incarcerated in an inflammatory cyclitic membrane. Enucleation four months after injury. A.I.P. Negative 97296.

planted and behaved as inactive foreign bodies, unlike conjunctival epithelium (fig. 16) which finds the interior of the globe a fertile field for growth.

Some of the eyes in this series were removed because of the severity of the initial injury and before secondary pathologic changes had appeared. In these, partial evisceration, disorganization, massive intraocular hemorrhage (fig. 17), and collapse of the globe were the kinds of damage which led to early enucleation. All stages of organ-

Fig. 17 (Wilder). Expulsive hemorrhage following a penetrating wound at the limbus by a copper fragment retained until enucleation nine days after a booby trap explosion. A.I.P. Negative 84004.

Fig. 18 (Wilder). Beginning organization of hemorrhage in the track of a lead foreign body, extending from the corneal wound of entrance to the chorioretinal wound of lodgement at macula. Enucleation 16 days after injury from gun discharge. Magnet extraction attempted on the day of injury. A.I.P. Negative 83982.

izing hemorrhage were present in eyes removed after longer postoperative periods (figs. 18 and 19).

Abscesses were frequently seen around the sites of foreign bodies of various types, almost invariably around vegetable matter (fig. 20). In the vitreous chamber, the subsequent formation of a pyogenic membrane resulted in retinal detachment (fig. 21) and phthisis bulbi (fig. 22). Glaucoma, a less frequent sequela of intraocular foreign body, was present in association with siderosis bulbi. It also occurred occasionally when the missle lodged in the anterior chamber and the inflammatory process was confined to the anterior segment (fig. 23). Foreign-body granulomas appeared around all kinds of foreign bodies, but the formation

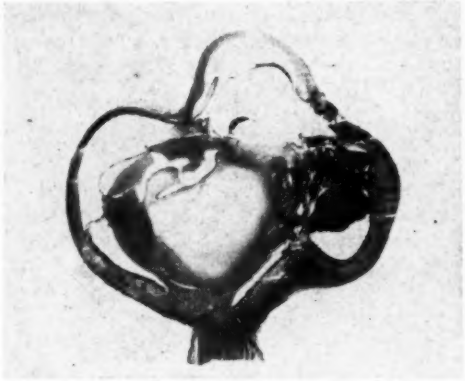

Fig. 19 (Wilder). Organized hemorrhage forming a traction band between the scar of entrance at the limbus and the chorioretinal scar of lodgement of a lead foreign body. Cicatricial distortion of globe. Enucleation six months after injury. A.I.P. Negative 97836.

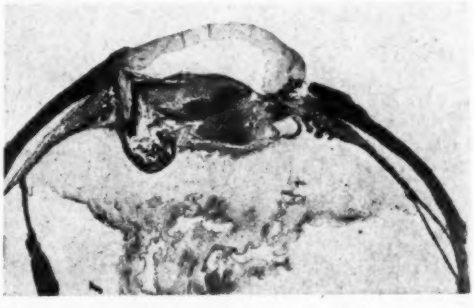

Fig. 20 (Wilder). Abscess around a vegetable fragment in the angle of the anterior chamber and in its track through the lens. Enucleation 24 days after injury. A.I.P. Negative 97293.

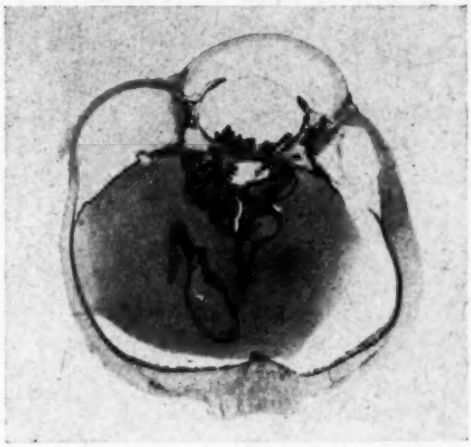

Fig. 21 (Wilder). Vitreous abscess at the site of a brass foreign body. Detachment of retina by a pyogenic cyclitic membrane. Early phthisis bulbi. Enucleation two months after injury. A.I.P. Negative 99314.

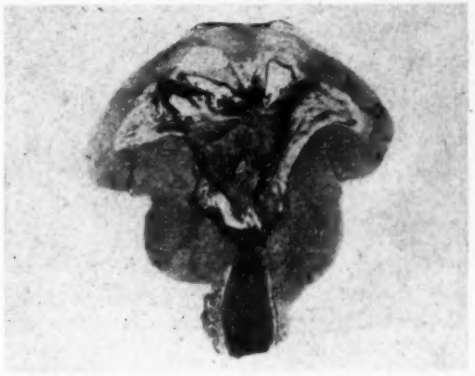

Fig. 22 (Wilder). Phthisis bulbi following lodgement of vegetable matter in the vitreous chamber 11 years before enucleation. A.I.P. Negative 99316.

of giant cells seemed to be stimulated particularly by eyelashes (fig. 24) and vegetable matter (fig. 25). In some instances large syncitial cells were arranged around implanted hairs in such a way as to suggest proliferated sheath cells. Phagocytized melanin granules were usually present in them, however, confirming the opinion that they were more probably true histiocytic giant cells. The tendency of vegetable matter to stimulate a giant-cell reaction is evidenced in a case in which cotton fibers and carbon particles were introduced into the vitreous chamber by a dynamite explosion. Endophthalmitis developed and the eye

Fig. 23 (Wilder). Chronic glaucoma following lodgement of a vegetable matter in the anterior chamber 14 years before enucleation. A.I.P. Negative 99313.

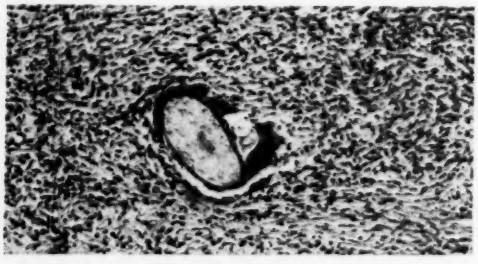

Fig. 24 (Wilder). Giant cell reaction to cilium in an inflammatory cyclitic membrane. Enucleation five weeks after injury. A.I.P. Negative 97363.

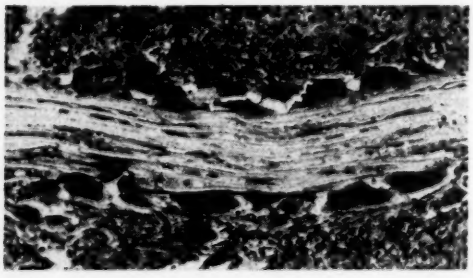

Fig. 25 (Wilder). Giant cell reaction to vegetable matter in an inflammatory cyclitic membrane. Enucleation two months after injury. A.I.P. Negative 97288.

was enucleated six weeks following injury. Incarcerated in an inflammatory cyclitic membrane, the cotton fibers (fig. 26) were all surrounded by giant cells, whereas none appeared around the carbon particles (fig. 27).

Sympathetic uveitis developed in five patients with retained intraocular foreign bodies. In one instance, lead (fig. 28) had been introduced in combat two months before enucleation and in another, wood (figs. 29 and 30) had been introduced in a preinduction injury 36 years before enucleation. In the latter, following the old corneal perforation, the eye retained a small amount

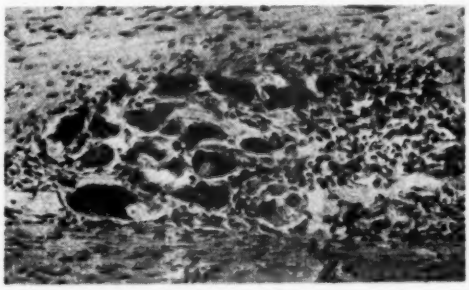

Fig. 26 (Wilder). Giant cell reaction to cotton fibers in an inflammatory cyclitic membrane. Enucleation six weeks after dynamite explosion. A.I.P. Negative 99317.

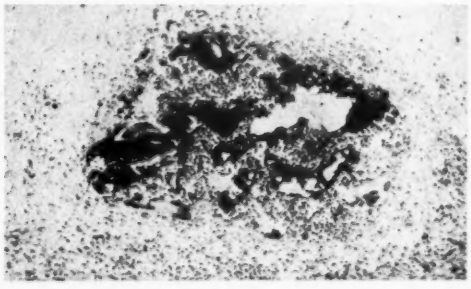

Fig. 27 (Wilder). Same eye as in Fig. 26. No giant cell reaction to carbon particles in the cyclitic membrane. A.I.P. Negative 99320.

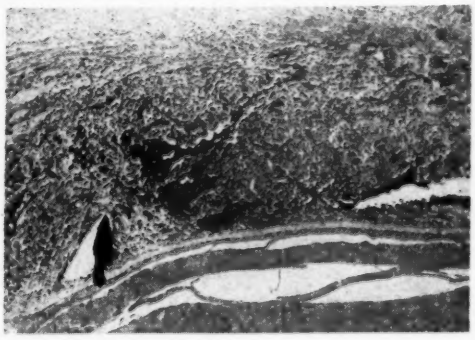

Fig. 28 (Wilder). Sympathetic uveitis evidenced by granulomatous inflammation of the uveal tract involving the iris. Lead foreign body in the iris. Posterior synechia. Enucleation two months after injury. A.I.P. Negative 97294.

of vision until three months before enucleation, when it became inflamed and blind. Uveitis appeared in the sympathizing eye one month before enucleation. One eye retained a fragment of glass until enucleation 1½ months after injury. One was removed three months following the introduction of an un-

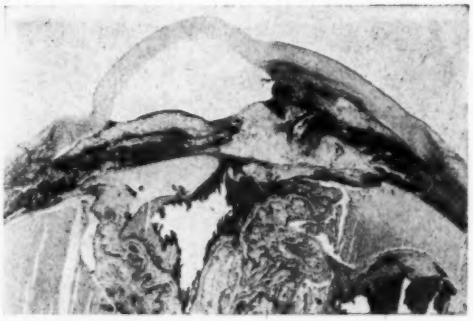

Fig. 29 (Wilder). Recent sympathetic uveitis in an eye with wood foreign bodies retained 36 years after a penetrating wound through the cornea. A.I.P. Negative 97358.

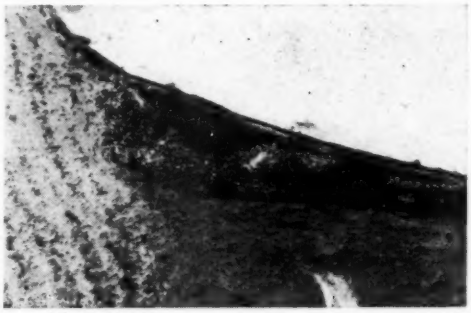

Fig. 30 (Wilder). Same eye as in Fig. 29. Choroid at optic disc. Sympathetic uveitis. A.I.P. Negative 97362.

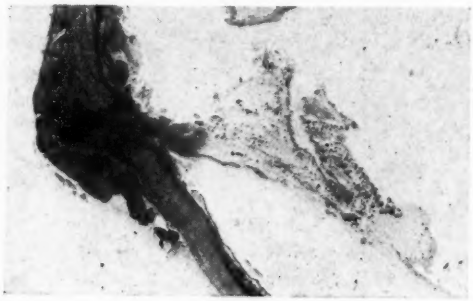

Fig. 31 (Wilder). Colonies of cocci in a vitreous abscess following a penetration of the sclera by a copper fragment which lodged in the vitreous. Enucleation two weeks after the explosion of a dynamite cap. A.I.P. Negative 97291.

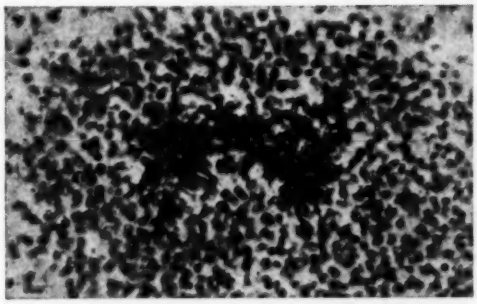

Fig. 32 (Wilder). Colony of unidentified fungi in a vitreous abscess. A minute nonmagnetic foreign body was found in the abscess on gross examination. Enucleation followed penetrating wound sustained in Italy. Duration unknown. A.I.P. Negative 97283.

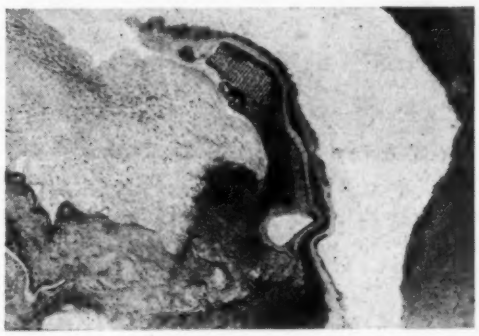

Fig. 33 (Wilder). Wood fragment prolapsed with retina through corneal wound in purulent panophthalmitis. Enucleation eight days after injury. A.I.P. Negative 97355.

identified nonmagnetic foreign body. In the fifth case the time interval between injury and enucleation was not given, but about one week after injury, severe uveitis was reported to have developed in the sympathizing eye. The foreign body was magnetic.

A variety of infections followed introduction of foreign material into the eye. Masses of cocci (fig. 31), bacilli, and unidentified fungi (fig. 32) were sometimes seen in vitreous abscesses. Saprophytic fungi were frequently found in and around wood particles (figs. 33, 34, and 35).

Comment

A wide variety of foreign bodies was found in eyes of soldiers following injuries in training and combat. Of these approximately 37 percent were magnetic, 62 percent

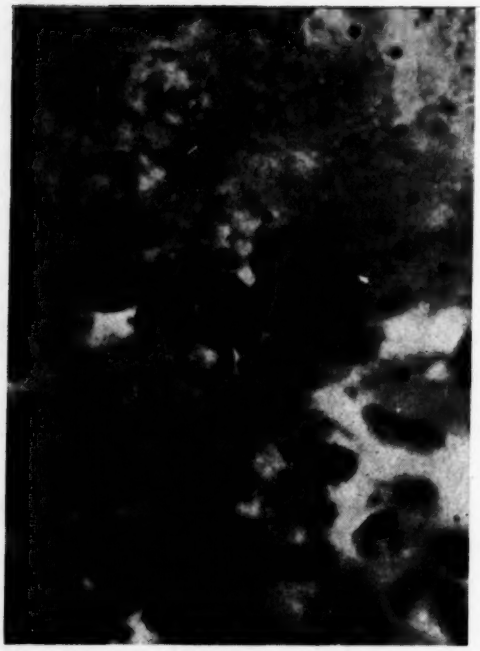

Fig. 34 (Wilder). Same eye as in Fig. 33. Brown yeast-like organisms, probably saprophytic, in vitreous abscess. A.I.P. Negative 99240.

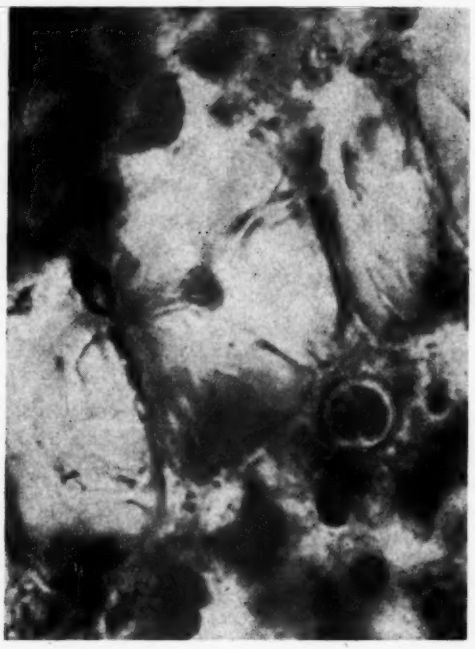

Fig. 35 (Wilder). Same eye as in Figures 33 and 34. Saprophytic fungi associated with wood particle in vitreous abscess. Double contoured cell and mycelium. A.I.P. Negative 99241.

nonmagnetic, and 1 percent mixed. This apparently confirms previous observations[1, 2, 3] that in military injuries nonmagnetic intraocular foreign bodies predominate. They do, undoubtedly, in eyes submitted for pathologic examination, but many instances of successful magnet extraction do not come to the pathologist's attention; and, even in eyes that are enucleated, nonmagnetic foreign bodies are often secondary to ferrous missiles which may have been extracted. The infections and reactions associated with the different types of foreign bodies ran the gamut from staphylococcus infection, resulting in purulent panophthalmitis, to the mild reactive inflammation associated with siderosis bulbi and intraocular hemorrhage without infection. The formation of giant cells was particularly abundant around vegetable matter and eyelashes. Of particular interest were five cases of sympathetic uveitis associated with retained nonmagnetic material in four eyes and with a retained ferrous foreign body in one. Phthisis bulbi was a common end result, and glaucoma was comparatively rare, as in any series of penetrating wounds.

I wish to thank Lawrence P. Ambrogi and Eleanor V. Paul for X-ray studies, careful gross examinations, and magnet tests, as well as for their preparation of sections which always included the foreign body sites.

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THE SURGICAL CORRECTION OF PARESIS OF THE SUPERIOR OBLIQUE*

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It is well recognized that paresis of the superior oblique is, with the exception of paralysis of the external rectus, the most common paresis of any individual ocular muscle. In the past three quarters of a century, cases of underaction of this muscle have been reported with increasing frequency, but until 1934 when Wheeler[1] reported upon his procedure for the advancement of the tendon, the surgical relief of paresis of the superior oblique was confined to procedures upon its yoke or direct antagonist muscles, with occasionally a recession of the superior rectus, or a resection of the inferior rectus of the same eye. Even since Wheeler's report, the vast majority of attempts at correction of the condition have dealt with the yoke or direct antagonist muscles and, only in rare instances, has the tendon of the superior oblique been operated upon. Whether or not this is due to the deeply ingrained belief of most ophthalmologists that it is dangerous to operate upon the superior oblique, I cannot say, but it is in an attempt to dispel this fear that this study and case reports are presented.

HISTORY

The malposition of the eyes which constitutes strabismus has been recognized from the earliest period of medical history, but not even the anatomy of the muscles concerned in ocular movements was understood with approximate accuracy until studied by Fallopius[2] in the 16th century. In the middle of the 18th century John Taylor[3] claimed to straighten cross-eyes by operation. Sir Charles Bell,[4] in 1823, divided the recti and oblique muscles of the eyes of lower animals and concluded that the former had to do with the voluntary and the latter with the involuntary movements of the eye. In 1827 Anthony White[5] suggested that by cutting the ocular muscles one could correct strabismus but the suggestion was not acted upon until Dieffenbach[6] operated.

Meanwhile, the treatment relied upon for squint was the wearing of a mask with openings through which the eyes could see only when properly directed, as suggested by Paulus Aegineta[7] in the 7th century; or the occlusion of the fixing eye proposed by Erasmus Darwin,[8] in 1778. In 1831 Strohmeyer[9] performed a tenotomy on the tendo achillis and followed up his first partial success by operating on muscles and tendons in all parts of the body. In 1838 Strohmeyer did a "myotomy for strabismus" on a cadaver. The era of operation upon the eye muscles may be said to begin with the announcement of Dieffenbach (1839) that he had obtained a perfect result in a case of convergent strabismus by division of the internal rectus muscle.

ANATOMY

The muscle arises by a short tendon just anterior and medial to the optic foramen, from which it is separated by the medial rectus, in the angle between the zonule of Zinn and the periorbital fascia. The belly of the muscle runs forward along the nasal wall of the orbit, close to the internal rectus but separated from the latter by the nasociliary nerve and ophthalmic artery and crossed by their ethmoidal branches. A few mm. behind the

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orbital margin at its upper nasal angle the muscle becomes tendinous and passes through a cartilaginous ring, the pulley or trochlea. This is the physiologic origin of the muscle. The tendon then bends backward, laterally and slightly downward, pierces Tenon's capsule and passes beneath the superior rectus to be inserted into the sclera in the superior posterior lateral quadrant of that structure. The tendon is enveloped by a prolongation of Tenon's capsule from its insertion to the pulley, and is, on the average, 20 mm. in length.

Embryologically Whitnall[10] states that the reflected portion of the muscle, that is, the tendon, from the trochlea to the insertion in the sclera, is fleshy in most mammals, and in the lower vertebrates the whole muscle is represented by the reflected portion only, this arising from the medial angle of the orbit near the origin of the inferior oblique.

The line of insertion of the tendon crosses the vertical meridian of the globe obliquely and is curved with a backward directed convexity. In the anterior part the tendon is narrow and almost rounded but near the insertion it spreads out in a fan-shaped insertion.

The distance from the corneal margin to where the tendon crosses the vertical meridian has been measured by Fuchs[11] who found it to be from 16 to 17.9 mm.. and by Weiss[12] who found the anterior edge of the obliquely placed line of the tendon to lie 13.85 mm. from the cornea and 14.66 mm. from the optic nerve, while the posterior end was 18.8 mm. from the cornea and 7.56 mm. from the nerve.

Fortin[13] has stated that the insertion of the superior oblique into the sclera lies just above the macular region and attributes many of the cases of retinal detachment in the macular area to the pull of the tendon of the superior and inferior obliques upon the sclera in this region.

The trochlear or fourth nerve crosses over the direct portion of the muscle from the medial side to enter its substance at about 12 mm. from the origin while the blood supply is derived from the superior muscular branch of the ophthalmic artery.

The trochlea or pulley of the superior oblique consists of a finely grooved and curved plate of hyaline cartilage which is attached to the periosteum of the frontal bone by fine strands of fibrous tissue, thus forming a tube through which the tendon slides. The tendon, as it passes through the trochlea, is invested by very lax areolar tissue with a poorly formed endothelial lining but according to Macalister[14] there is hardly a true synovial membrane lining the trochlea. The elongation of Tenon's capsule which envelops the tendon up to the pulley is fixed to and terminates at the latter. Embryologically the pulley was a specialized part of the sheath of the muscle at its origin, which was situated near the orbital margin, but the need for a longer muscle belly in mammals has led to the development of the pulley and the retrogression of the origin backward toward the apex of the orbit.[10]

Maddox[15] has suggested that the reason why the superior oblique has a pulley is that since the speed with which a muscle's point of insertion moves is proportional to its length, it is necessary for the superior oblique, the virtual origin of which as regards its action is from the pulley, to have a long, fleshy, reflected portion extending to the back of the orbit if it is to keep pace with the movements of the recti.

Whitnall[10] suggests that as regards this advantage over the inferior oblique, the reason may be that prolonged looking downward is more important for daily work than looking upward and, therefore, the excursions of the eye due to the superior oblique are more amply provided for than those of the inferior oblique.

Physiology

The principal action of the superior oblique is to turn the eyeball down and

inward. There are two subsidiary actions of the muscle, the first being abduction of the eyeball and the second intorsion of the upper part of the vertical meridian. The main action of the muscle increases when the eye is turned inward and becomes practically nil when the eye is abducted. Thus, it is seen that when the globe is turned medially by the internal rectus and then depression is desired, the superior oblique of that eye exerts the strongest force upon the eye that is possible. When the eyeball is turned outward, the tendon of the superior oblique is situated so that it is almost at right angles to the anterior posterior axis of the globe and consequently its main action, that of depression of the bulb, is reduced to zero. At the same time, however, the secondary actions of the muscle increase when the globe is turned outward, because the tendon of the muscle lies in such a position that its effect on intorsion and abduction is greatest. Conversely, when the globe is adducted, the effect of the superior oblique on abduction and intorsion is markedly decreased.

Another function of the superior oblique, combined with the inferior oblique, has been advanced by various authors among whom are Theobald,[16] Fuchs,[17] Duane,[18] Jackson,[19] and Bielschowsky.[20] This is that the two obliques act in opposition to the four recti when they tend to pull the globe back into the orbit. Samuel Theobald[16] advanced the theory that the principal function of the obliques, assuming that they did resist the action of the four recti in pulling the globe back into the orbit, was to cause the eye to rotate, "with precision, about a fixed center, a provision so essential to accurate vision, and, especially, to perfect binocular vision."

That the physiologic function of the superior oblique has been interfered with is shown in two reports, one by Peter,[21] in 1934, and the other by Sanford Gifford,[22] in 1942, in which transplantation of the tendon of the muscle to the tendon of the internal rectus was performed in cases of paralysis of the third nerve. In each case the eye, in the primary position, was rotated outward, and in each case the external rectus was recessed prior to the transplantation of the superior oblique. Both authors reported that abduction was limited by the total procedure, but failed to mention that the eye could be adducted to any degree beyond the midline as a result of the operative interference. This leads one to the speculation that the cosmetic improvement in these cases was due to the recession of the external rectus and not to the transplantation of the superior oblique.

In support of the theory of the transference of function in the transplantation of the extraocular muscles is the work of Olmstead, Margutti, and Yanagisawa,[23] in 1936, who conducted experiments on the extraocular muscles of dogs, cats, and monkeys in which various muscles were transposed with a view to finding out whether coördinated movements after operation might not be the result of the interaction of the undisturbed muscles, and, if real transference of function does occur, whether there is possible a brief period of readjustment, or learning, which would indicate that the original conducting pathways are predetermined, and new ones must be laid down to meet the changed conditions.

After the series of experiments in which muscles were tenotomized and other operations in which muscles were transplanted, these authors came to the conclusion that the first movements of eyes of animals coming out of anesthesia after operation were in the direction that one would expect if impulses to muscles were coming out over the usual pathways, but gradual adjustment to the new situation took place with the lapse of time.

Leinfelder and Black,[24] in 1942, studied the effect of the superior oblique, along with the other extraocular muscles, in re-

covery of muscular function following tenotomy of the recti muscles. These authors outlined a series of experiments in which the physiologic effects of the superior oblique could be controlled and interpreted while effectual transposition of the muscles was being accomplished in successive operations.

These operations consisted in tenotomizing and resecting the superior oblique in monkeys, either before or after other transposition operations were done. These experiments showed that interference with coördinated rotations did not occur as long as two muscles retained their normal anatomic and physiologic relationships. This is illustrated by the recovery of coördination when only the four recti were transposed; whereas, failure of recovery occurred when the superior oblique was also tenotomized.

The authors conclude that the two intact oblique muscles might be considered capable of giving the proprioceptive impulses necessary for recovery of associated movement; whereas, only one muscle is inadequate. In other words, the superior oblique has no special function in giving the necessary proprioceptive impulses when it is acting alone.

It would seem then that the basic physiology of the superior oblique reverts to the action of depression of the globe as its main function with intorsion and abduction as the secondary functions. It is true that there may be other functions of the muscle, but these are at present purely within the realm of speculation, and it is with the obvious functions of the muscle and the alterations thereof that this paper is concerned. There is here no thought of altering these functions, but merely of the most logical manner of increasing them in cases of paresis of the muscle.

Pathologic physiology

Bielschowsky[25] has stated that, if, in a case of orthophoria, a single muscle becomes paretic, deviation in the primary position is always present, being latent or manifest according to the intensity of the paresis and the function of the paretic muscle. In a case of heterophoria the paretic deviation is modified by the anomalous position of rest. In any case of paresis the position of rest must be altered by the loss of tonus of the paretic muscle and the intact tonus of the other muscles. If this is the only reason for deviation, orthophoria will be restored as soon as the paresis is cured. This cannot happen if the deviation is caused, in addition, by the development of a secondary contracture of the antagonist of the paretic muscle, due to a change in its structure. In any case of typical paresis of a single muscle the angle of deviation increases or decreases according to whether the movement of the eye is in the direction of action of the paretic muscle or of its antagonist.

Diagnosis of muscular palsy can be made if movement of the paretic eye in a certain direction is limited, but it must be remembered that even a paretic muscle can bring the eye to the normal limit by means of an excessive innervation. In many cases of palsy an anomalous position of the head is a characteristic sign, which is often explained as an endeavor to make up for the function of the paralyzed or weakened muscle by turning the head in the direction in which the eye muscle had been acting before it was paralyzed.

Bielschowsky has modified this rule to the effect that the patient chooses the least inconvenient position of the head by which the paretic muscle is sufficiently disburdened so that binocular single vision can be obtained. In cases of trochlear nerve palsy the head is tilted toward the shoulder of the sound side and at the same time rotated on the vertical axis so that the paretic eye is rotated outward.

Trochlear nerve paralysis is the most frequent type of paralysis of a single vertically acting muscle and the most striking

sign of this is habitual torticollis. The ocular origin of this kind of torticollis was first recognized by Cuignet.[26] He, however, could not explain the connection any more than could Landolt[27] in his paper on the subject.

Prior to Landolt, A. Nagel,[28] in 1871, had supposed that in cases of slight paresis of an elevator or depressor muscle, a vertical and rotary deviation would be caused by tilting the patient's head toward one side, a supposition based on the discovery that a parallel rotation of the eyes around the visual axis is produced by tilting the head toward the opposite side.

In a case of right trochlear nerve palsy if the head is tilted toward the right shoulder, there will arise a vestibular excitation of the vertical motor muscles which are able to produce a parallel rotary movement of the eyes to the left, the movement being produced in the left eye by the two inferior muscles and in the right eye by the two superior muscles. The paralyzed right superior oblique cannot compensate the elevating and adducting component of the superior rectus, from which a vertical and lateral deviation of the right visual line must result; whereas, in the normal person both the visual axes would be stationary.

If the head of the patient with the paretic right superior-oblique muscle is tilted toward the left side, both inferior muscles of the right eye and the superior muscles of the left receive the vestibular innervation to rotate the eyes around to the right. This movement can be performed without the coöperation of the paretic muscle. Consequently, no deviation of the visual axis will result. The more the sound muscles are burdened, the more favored will be the paretic muscle. The habitual tilting of the head in trochlear nerve palsy is thus understandable. If the head is tilted toward the shoulder of the sound side, the paretic superior oblique is favored and binocular single vision is obtained.

Bielschowsky[29] believes that in cases of "incurable" paralysis of the superior oblique a contraction of the inferior oblique muscle of the same eye prevents the decrease of the vertical deviation in looking upward as seen in typical cases, and finally the deviation becomes entirely independent of vertical movements.

Etiology

Many cases of superior-oblique paralysis have been reported in opthalmic literature, most of them having been one or two isolated cases. It is well recognized that next to paresis of the external rectus, paresis of the superior oblique is the most frequently occurring weakness of an individual muscle.

Snell,[30] in reporting some cases of traumatic paralysis of the superior oblique, in 1919, stated that approximately 60 percent of the cases of traumatic disturbances of the superior oblique make a satisfactory recovery wthin eight months. He held that the disturbances of this muscle due to indirect injury were most likely to recover, and even those due to direct injury where the muscle or its tendon has not been completely severed, have ceased to cause diplopia or annoying symptoms after a few months.

Sorsby,[31] in 1932, reported a case of paralysis of the superior oblique due to a knife wound, which penetrated the upper inner angle of the orbit. Savin,[32] in 1934, reported the case in which a boxer was struck in the face and immediately had a diplopia which proved to be consistent with paresis of the superior oblique. This persisted for months and was finally relieved to some extent by a recession of the homolateral superior rectus.

Probably the most comprehensive single report on the etiology of paresis of the superior oblique is that of Bielschowsky,[29] in 1939, who holds that there has been an undeniable increase in the percentage of cases of paralysis of the trochlear nerve. He does not feel that this in-

crease can be attributed to an improvement in methods of investigation, since all the cases included in the statistics he reports had been examined solely by him. Up to 1908 all cases of trochlear nerve paralysis amounted to 10 percent of all cases of paralysis of the extraocular muscles, or not quite half as many as the the cases of paralysis of the abducens nerve. From 1908 to 1932, the percentage of cases of superior-oblique paralysis increased to 20 percent and that of abducens-nerve paralysis remained the same as before or about 25 percent. This author believes that the striking increase is due to the introduction of Killian's operation and similar radical operations on the frontal sinus. In support of this belief, the author reports 15 out of 80 cases seen by him between 1923 and 1932 which had definitely been caused by operations on the frontal sinus.

All cases of paralysis of the extraocular muscles will show approximately the same relative etiology. In other words, there is no specific cause which singles out one individual muscle. It is the general consensus that trauma is the most frequent cause of these disturbances in ocular motility. Syphilis is thought to play a prominent part and next in frequency are cases of congenital origin and those due to encephalitis. Two rare cases are mentioned by Bielschowsky of paralysis of the abducens nerve due to severe loss of blood. These recovered spontaneously within a year's time. Pareses of various extraocular muscles have been reported as a complication of diabetes.

In support of the theory of Bielschowsky[29] mentioned above that the striking increase in pareses of the superior oblique is due to the great increase in radical frontal sinus operations, one has but to consider the approach to the frontal sinus in a radical operation, as seen in various illustrations in texts and monographs on exposure of this air space, to wonder why there are not 100-percent pareses of the superior oblique in this operation.[33, 34] It can be granted that the operation is necessary to relieve a condition that is amenable to nothing else but radical surgery, and that the resultant paresis of the superior oblique generally clears up spontaneously, but at that point we arrive at an impasse. We must agree that the radical operation for frontal sinusitis is necessary in some cases and that the best approach so far devised is that most commonly in use by the majority of otolaryngologists. The pulley of the superior oblique can easily be damaged by this approach either by a slip of the chisel, an overbite of the rongeur or simply damaged by the retractor. The ophthalmologist should regard this operation as a necessary evil, and, in those cases in which spontaneous recovery does not occur, he should be prepared to correct as far as possible the distressing diplopia that results from such surgery.

Paresis of the superior oblique

Signs and symptoms

As has been mentioned previously, the most striking sign of paresis of the superior oblique is habitual torticollis with the head most frequently tilted toward the opposite side from that of the paretic muscle. There is, of course, a hypertropia which increases when the eye is carried into the field of action of the superior oblique. Subjectively, there is the vertical diplopia increasing when the paretic eye is carried down and in, and, in some cases, a tilting of the images due to a disturbance in the torsion effect of the superior oblique. In addition to these symptoms many patients will have nausea, and even occasionally vomiting, particularly when an attempt is made to carry the head straight after an habitual torticollis has been established.

Surgical correction

The direct attack upon the superior-oblique muscle, either in paresis or over-

action, has been, for the great majority of ophthalmologists, something to be avoided at all costs. The principal objection seems to have been that the tendon of the muscle is too difficult of access.

Jackson[19] stated that because of its deep insertion, operative interference upon the tendon of the superior oblique near its insertion is not practicable. He advocated transplantation of the superior rectus for paresis of the superior oblique and, by his procedure, the tendon of the superior rectus was recessed and also placed slightly more temporally in order to exert more influence in causing intorsion of the globe. Landolt[35] recommended advancement combined with resection of the inferior rectus in cases of underaction of the superior oblique of the same eye.

Snell[30] held that when operation had been decided upon in paresis of the superior oblique the choice was between a tenotomy of the superior rectus of the same side and a tenotomy of the inferior oblique of the same side.

Banister[36] advocated advancing or tucking the inferior rectus of the affected eye sufficiently to bring the eye on a level with the other eye in the primary position. Savin[32] reported a case of diplopia due to paresis of the superior oblique relieved by recession of the superior rectus of the same eye.

Jaensch,[37] in reporting on a case of binocular trochlear paralysis, advocated recession of the inferior recti as the first step and this was to be followed by myectomy of the inferior oblique, if necessary.

White,[38] in discussing paralysis of the superior oblique, gives the following criteria for operative correction: if the hyperphoria is moderate, a recession or tenotomy of the inferior rectus of the opposite eye gives good results. If there is a secondary contracture of the inferior oblique of the same eye, causing a marked hyperphoria or hypertropia in the upper field, a tenotomy of the homolateral inferior oblique is indicated. If the vertical deviation is marked, White thinks it may be safer to tuck the superior oblique first, this to be followed by a tenotomy of the inferior oblique of the same eye, a recession of the inferior rectus of the opposite eye or both, depending on the subsequent measurements.

Spaeth[42] recommends recession of the contralateral inferior rectus in paresis of the superior oblique, while Dunnington,[43] in 1931, in quoting Duane's table of choice, advocated tenotomy of the inferior oblique of the same eye or recession of the inferior rectus of the opposite eye.

Jackson,[39] Peter,[21] Wiener,[40] Sanford Gifford,[22] and Wegner[41] have all reported cases of transplantation of the tendon of the superior oblique to the insertion of the internal rectus in cases of oculomotor palsy, holding that the tendon of the oblique when transplanted in this condition tends to act as an internal rotator.

As far as can be determined, surgery on the muscle itself in cases of disturbances of motility due to the superior oblique has been described by four authors. Hughes and Bogart,[44] in 1942, described their method for recession of the trochlea in cases of overaction of the superior oblique. Axenfeld[45] reported myotomy of the inferior oblique followed by "extirpation" of the trochlea for cosmetic purposes by exclusion of both obliques. Van der Hoeve[46] stated that tenotomy of the superior oblique was seldom performed, but that according to Czermak and Elschnig one method is to progress from the conjunctiva after detachment of the superior rectus. Wheeler[1] reported his method for advancement of the underacting superior oblique, in 1934.

My interest in the surgical correction of paresis of the superior oblique was stimulated by the report of Wheeler[1] on his advancement procedure for underaction of the muscle. The method which I now use is essentially a modification of Wheeler's procedure and was devised simply because it seemed to offer a little surer

method of achieving the desired result. In the six cases of paresis of the superior oblique which are reported in this paper, seven operations were performed. Wheeler's method was followed in the first three operations. After failure with this procedure, the writer's modification was introduced in the second operation on the third case and followed in the last three cases.

Resection of the tendon

Because of the posterior location of the insertion of the tendon of the superior oblique, general anesthesia is almost indispensable and, in the six cases reported, intravenous sodium pentothal was found to be an ideal anesthetic.

A traction suture is placed through the sclera just above the upper limbus and the eye turned well down by this means. The conjunctiva is incised over the insertion of the superior-rectus tendon. Tenon's capsule is opened and the rectus delivered on a muscle hook (fig. 1).

This muscle is freed of its subconjunctival attachments by means of sharp dissection with scissors and a double-armed 4-0 plain-catgut suture is passed through the tendon close to the insertion. The tendon is then cut between the suture and the globe and the superior rectus is retracted and freed by blunt dissection from the underlying tendon of the superior oblique. An additional traction suture may be placed through the stump of the rectus (fig. 2).

A muscle hook is passed backward over the globe and under the tendon of the superior oblique which it engages. Another double-armed 4-0 plain-catgut suture is passed through the tendon at a variable distance from its insertion, depending upon the amount of correction that is necessary (fig. 3).

The tendon is cut just distal to the suture and the free portion of the tendon then cut free from the globe at its insertion (fig. 4). The two ends of the suture are passed through the stump of the tendon and tied (figs. 5 and 6). The superior rectus is sutured back into position and the conjunctiva closed with plain catgut or fine silk.

The advantages of this modification of Wheeler's operation are two. In the first place, in the original procedure described by Wheeler, the tendon of the superior oblique is tucked without providing for the apposition of raw surfaces and, after one failure with this method, in which the adhesions failed to form at all, the writer came to the conclusion that if two raw surfaces were brought in apposition the chances of a firm union would be much better. Subsequent operations have proved this to be true. In the second place, the work of Fortin[13, 47] and Genet[48] and others has shown that the two oblique muscles of the eye have their insertions in the sclera close to, or in, the macular region. In view of this, it would seem that the insertion of sutures through the sclera in this region would definitely tend to produce more trauma to the macular region than the introduction of sutures merely through the stump of the original insertion into the sclera.

A word should be interjected at this point as to the ease or difficulty in operating upon the tendon of the superior oblique. The principal objection to surgery upon the tendon itself has been voiced by von Graefe[49] and Jackson,[19] both of whom stated, in effect, that the tendon was too difficult of access to permit surgery upon it. Wheeler,[1] himself, in describing his operation states, "It cannot be said that the operation is convenient or easy, but technically it is entirely possible and reasonable."

With the above premises the writer must disagree. The approach to the tendon of the superior oblique is certainly not too difficult for any competent ophthalmic surgeon and once the tendon is exposed, the remainder of the procedure is not complicated nor technically difficult.

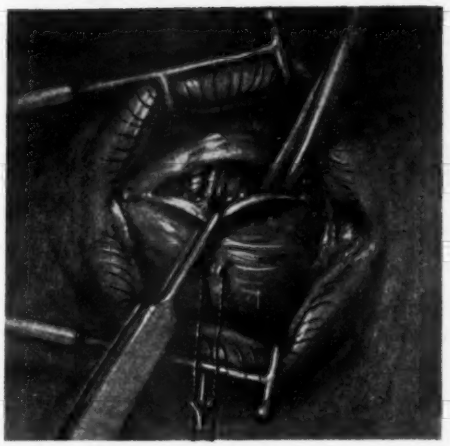

Fig. 1 (McGuire). Exposure of superior rectus.

Fig. 2 (McGuire). Exposure of tendon of superior oblique.

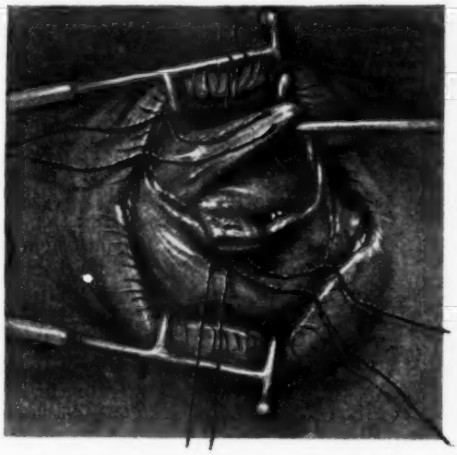

Fig. 3 (McGuire). Suture through tendon of superior oblique.

Fig. 4 (McGuire). Tendon of superior oblique cut and distal portion excised at insertion.

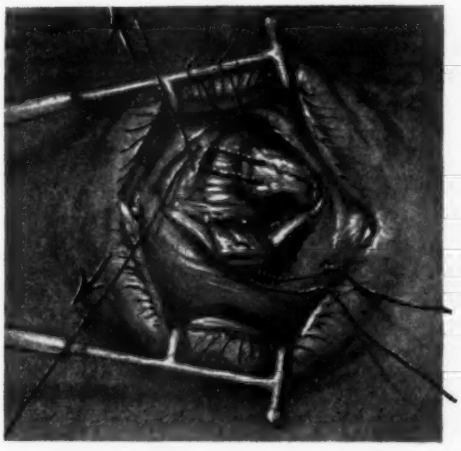

Fig. 5 (McGuire). Suture passed through stump of superior oblique.

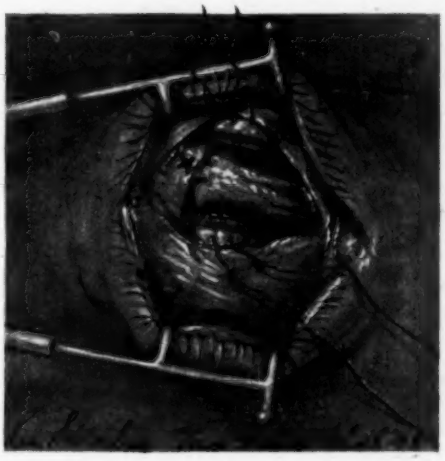

Fig. 6 (McGuire). Shortened tendon of superior oblique in position.

No definite rules can be given as to the amount of tendon to be resected, but an immediate overcorrection is to be desired. An estimate of the amount of tendon to be resected can be obtained by having the globe turned strongly down and in by means of the traction suture and, with the eye in this position, grasping the tendon with a muscle forceps at various distances from the insertion and then doubling the tendon over upon itself by carrying the forceps toward the insertion. If the forceps are placed upon the tendon in such a position that when they are moved over to the insertion the tendon is fairly tight, the ideal amount of tendon to be resected will have been determined. The suture should then be placed in the tendon at this point and the tendon between the suture and the insertion resected.

Postoperatively there is generally more reaction following this procedure than is usual with operation upon the recti muscles and this is no doubt due, in large measure, to the fact that the insertion of the tendon is so far posterior on the globe. The reaction will subside markedly within a week or 10 days and it has not been found necessary to do more than firmly bandage the operated eye at any time.

Case reports

Case 1. M. J. G., aged 34 years, a Lieutenant in the USNR, was admitted to the hospital under the general diagnosis of strabismus. His chief complaint was diplopia when the head was held straight. Aside from the usual childhood diseases with uneventful recoveries, the past history was entirely irrelevant. There was no history of head injury or other trauma. This officer stated that he had really never noticed diplopia until he had entered the service, but that he had always tilted his head toward his left shoulder. On entry upon active duty, he felt that he was conspicuous because he was in uniform and had a head tilt. He then determined to straighten his head in order to appear more natural. However, he found on attempting this that a marked diplopia was produced and he became so nauseated that at times vomiting occurred. He was consequently admitted to the hospital for treatment and disposition.

Physical examination on admission was entirely negative with the exception of the eyes. Laboratory studies revealed nothing of significance. Vision in each eye was 20/20. The media were clear and the fundi and intraocular pressure were normal. Muscle-balance tests by the screen and parallax method of Duane produced the following findings: Right hypertropia of 50 prism diopters and esotropia of 10 p.d. for both distance and near in the primary position. In the cardinal directions of gaze for near the findings in prism diopters were: eyes up and right RHT 20, ST 10; eyes right RHT 30, ST 10; eyes down and right RHT 30, ST 10; eyes up and left RHT 40, ST 5; eyes left RHT 50, ST 10; eyes down and left RHT 54, ST 10. There was a corresponding vertical diplopia, which increased when the eyes were carried into the field of action of the right superior oblique, and it was noted that this officer carried his head habitually tilted toward the left shoulder. On the basis of these findings, a diagnosis was made of paresis of the right superior oblique of congenital origin. It was felt that the hypertropia was the primary difficulty and that the esotropia would not bother him if the vertical deviation was corrected.

Under intravenous sodium pentothal anesthesia an advancement of the tendon of the right superior oblique was performed according to the technique described by Wheeler. Postoperatively there was moderate reaction, but this subsided markedly within 10 days. Three weeks after operation muscle balance was right hypertropia 14 and esotropia 10 prism diopters for both distance and near in the primary position. Additional measurements were: U&R RHT 0, EST 10; R RHT 10, EST 10; D&R RHT 10, EST 10; U&L RHT 10 EST 10; L RHT 15, EST 10; D&L RHT 22, EST 10. There was no further subjective diplopia although a vertical diplopia could still be elicited by the usual methods. The head tilt was markedly improved, but there was still some present to take care of the relatively slight imbalance which remained. Six months after operation the results were essentially the same as noted three weeks postoperatively. It is of some interest to note that this officer, following his operation, went to sea on an aircraft carrier and that as late as Christmas, 1945, stated in a letter to me that he had had no further symptoms and that he was very grateful for the operative result.

Case 2. H. M. W., aged 19 years, was admitted to the hospital with chief complaint of diplopia —duration lifetime. The patient stated that there had been a vertical diplopia present as long as he could remember, but that he could overcome this by tilting his head. He did not remember when he first discovered this. The past history was irrelevant and the physical examination and laboratory routine were negative with the exception of the eyes. Vision was 20/20 in each eye. The only significant finding in the examination of the eyes was in the muscle balance. There was a

right hypertropia for both distance and near of 14 prism diopters. Other measurements were: U&R orthophoria; R RHT 4; D&R RHT 8; U&L RHT 16; L RHT 20; D&L RHT 25. There was a corresponding vertical diplopia increasing in eyes down and left and a head tilt of moderate degree toward the left shoulder. A diagnosis of paresis of the right superior oblique was made and an advancement of the tendon by the Wheeler method was performed under intravenous anesthesia. Seven weeks postoperatively there was a residual right hypertropia of 4 prism diopters for both distance and near in the primary position, RHT 4 in eyes left and RHT 7 in eyes down and left. In the remainder of the cardinal positions orthophoria prevailed. The remaining vertical imbalance was compensated for by a slight head tilt, but this was not nearly so marked as the tilt prior to operation. Subjectively the patient felt much improved.

Case 3. J. R. F., aged 26 years, was admitted to the hospital with a diagnosis of "paralysis of ocular muscle." This young man stated that he was well until about a year before admission when he began to notice some blurring of vision. His general health had been excellent prior to the onset of this symptom and had remained so ever since. There was no complaint of diplopia. The past history and the laboratory studies were irrelevant. Neurologic and general physical examinations were negative with the exception of the eyes. Vision in each eye was 20/20; media were clear; fundi and intraocular pressure were normal. Muscle-balance tests gave the following results: right hypertropia of 7 prism diopters for both distance and near. Other measurements were: U&R orthophoria; R orthophoria; D&R RHT 5; U&L RHT 8; L RHT 14; D&L RHT 26. Although the patient had never complained of diplopia, there was a head tilt to the left and when the head was straightened there was a definite diplopia which corresponded to the muscle balance and increased in eyes down and left. A diagnosis of paresis of the right superior oblique was made.

Under sodium-pentothal anesthesia, a Wheeler advancement was carried out on the tendon of the right superior oblique. The postoperative course was uneventful, but upon examination two weeks after the operation, the muscle balance remained essentially unchanged, and it was apparent that nothing had been accomplished by the operation. It was believed that probably the suture between the tucked superior oblique and the sclera had pulled out before adhesion had taken place.

Five weeks after the first operation the original operative field was explored and the tendon of the superior oblique was found in its original position without any shortening effect whatever from the Wheeler procedure. At this time a resection of the tendon, as previously described, was carried out. The postoperative course was uneventful and the patient was shortly discharged to duty. Five months after operation this man was seen as an out patient for a final check at which time he stated that since his discharge from the hospital he had been doing office work without difficulty and that his symptoms had been relieved. There was an orthophoria in all cardinal directions of gaze except in eyes down and left where there remained a residual right hypertropia of 6 prism diopters.

Case 4. A. J. B., aged 26 years, was admitted with diagnosis of "insufficiency of ocular muscle." The main complaint was of diplopia when the head was held in the upright position. This condition had been present since childhood when the patient had fallen from a tree and struck his head on the roots, becoming unconscious for a short period. The general physical, neurologic and laboratory examinations were entirely negative as were X-ray studies of the skull. Ocular examination revealed vision to be 20/30 in the right eye corrected to 20/25 by a +0.5D. cyl. ax. 45°; and in the left eye to be 20/20. The media were clear, the fundi and tension normal. Muscle balance showed a right hypertropia of 10 prism diopters for distance and 12 for near in the primary position. Other measurements were U&R EX 5; R orthophoria; D&R RHT 8; U&L RHT 4, EX 5; L RHT 18; D&L RHT 32. There was a corresponding vertical diplopia increasing in the field of action of the right superior oblique, and a head tilt to the left. On the basis of the history and the muscle findings a diagnosis was made of paresis of the right superior oblique of traumatic origin. A resection of the tendon of the right superior oblique was then performed under sodium-pentothal anesthesia. Following his discharge from the hospital, the patient was followed in the outpatient department and three months after operation stated that he felt much relieved by the surgical procedure. At this time there was orthophoria for both distance and near in the primary position with a residual right hypertropia of 3 prism diopters in eyes left and of 9 p.d. in eyes down and left.

Case 5. J. W. F., aged 23 years, was admitted to the hospital with the diagnosis of "insufficiency of ocular muscle." He gave a history of having had double vision for as long as he could remember when the head was held straight, but this diplopia was eliminated by tilting the head to the right. General physical, X-ray and laboratory examinations were negative. Vision in each eye was 20/20 and the eyes were normal throughout with the exception of the extraocular muscles. In the primary position there was a left hypertropia of 14 prism diopters for both distance and near. Other measurements were: U&R LHT 5; R LHT 18; D&R LHT 27; U&L orthophoria; L LHT 8; D&L LHT 12. There was a corresponding vertical diplopia.

A diagnosis of paresis of the left superior oblique was made and under sodium-pentothal anesthesia a resection of the tendon of this muscle performed. The postoperative course was not unusual and one month after operation there was orthophoria for both distance and near in the primary position, and there was no vertical imbalance except in eyes down and right where there was a left hypertropia of 6 prism diopters.

Case 6. W. H. B., a man, aged 49 years, was admitted to the hospital with a diagnosis of frontal sinusitis. There was a history of intermittent bilateral frontal headaches of one month's duration. Clinical and X-ray examinations of the sinuses failed to reveal any pathologic changes. Laboratory studies were negative in all respects. The headaches increased rapidly in severity and on neurologic consultation it was felt that he probably had a subdural hematoma although there was no history of injury. A trephination was done on the skull and a fairly large subdural hematoma was found on the right side and evacuated. Following the operation, the patient began to complain of diplopia and then stated that this had been present for about the same length of time as his headaches but that he had neglected to mention the symptom. Approximately three weeks following the cranial operation the patient was seen in consultation by the eye service. The ocular examination was entirely negative with the exception of the muscle balance. There was a left hypertropia of 6 prism diopters for both distance and near in the primary position. Other measurements were: U&R LHT 8; R LHT 16; D&R LHT 23; U&L orthophoria; L orthophoria; D&L LHT 4. A diagnosis of paresis of the left superior oblique secondary to intracranial damage was made. Six weeks later the muscle findings were the same as measured above and it was believed that he was not going to regain the use of the superior oblique by spontaneous recovery. Consequently, a resection of the tendon of the left superior oblique was performed under sodium-pentothal anesthesia. The postoperative course was uneventful and two months later the diplopia had cleared up and there was orthophoria in all directions of gaze except in eyes down and right where there was a residual left hypertropia of 4 prism diopters.

Comment

Prior to 1934, when Wheeler[1] described his operation for advancement of the tendon of the superior oblique, paresis of this muscle had been treated surgically by one of three methods or a combination of these, that is, tenotomy of the inferior oblique of the same eye, recession of the inferior rectus of the opposite eye, and recession of the superior rectus of the same eye, with the first two methods being preferred by most operators. In rare instances the homolateral inferior rectus has been advanced. It would seem that none of these methods directly attacks the main problem. Complete paralysis of the superior oblique is rare and what the ophthalmologist is confronted with in the vast majority of cases of disturbance of motility of this muscle is an underaction, or a paresis, of the muscle. Granted that such is the case, the most logical primary surgical procedure in relieving the distressing symptoms of this condition is upon the paretic muscle itself. If such a procedure does not result in relief of the condition, the other methods may then be put into use. However, it is believed that with resection of the tendon of the superior oblique in paresis of this muscle as the primary step, secondary procedures will seldom be necessary and the patient will be saved the inconvenience, not to mention the expense, of several operations, the results of which cannot even be estimated in advance.

In regard to the oft propounded theory that the tendon of the superior oblique is too difficult of access to permit easy operation the writer firmly believes that the sooner this is forgotten, the better. With adequate exposure, by means of a traction suture pulling the eye down and in, the difficulties of access to the tendon are theoretical rather than real.

Summary

A brief historical review of the earliest surgery upon the extraocular muscles is presented.

The anatomy, physiology, pathologic physiology, and the etiology of paresis of the superior oblique is discussed.

The surgical correction of underaction of this muscle is reviewed and a modification of Wheeler's advancement operation is presented.

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NOTES, CASES, INSTRUMENTS

A NEW DRAINAGE OPERATION FOR THE RELIEF OF GLAUCOMA*

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Drainage operations in the field of glaucoma therapy meet only with incomplete and uncertain success. As an example one may consider the iridencleisis operation and its postoperative events. The scleral incision tends to close immediately and tightly, due to the inherent elasticity of the scleral tissue. The incision heals quickly and firmly because large areas of the mesodermal surfaces are in intimate contact. The included iris tends to undergo pressure atrophy as the result of sharp compression by the scleral lips. This event renders the operation practically valueless. A similar unfavorable outcome may be observed fairly frequently in all drainage operations now in vogue for the relief of ocular hypertension.

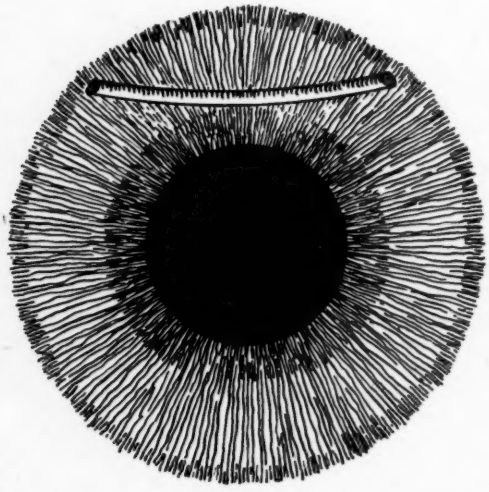

Fig. 1 (Lavine and Langenstrass). The corneal groove.

In reviewing the reasons for such failure, the following possibility was considered. All drainage operations form, or try to form, a passageway through connective tissues. As soon as the draining instrument (knife, keratome, spatula) is withdrawn, however, the draining canal collapses. The raw connective-tissue surfaces come in close contact. Fibroblasts proliferate. The channel starts to be closed almost immediately by scar formation. The glaucoma syndrome returns, because fluid production again exceeds fluid absorption.

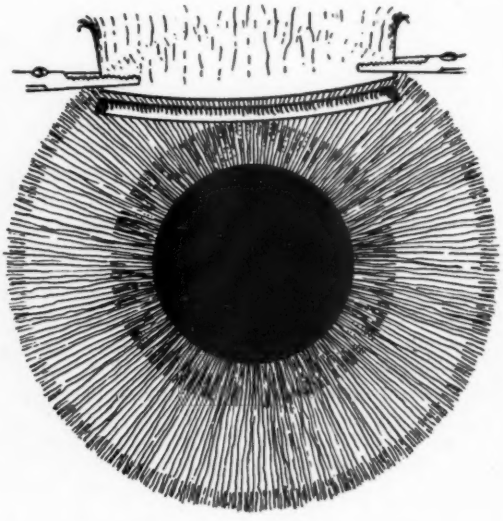

Fig. 2 (Lavine and Langenstrass). First conjunctival flap.

It would seem that a satisfactory drainage opening should ultimately be so constructed that its opposing walls receive a lining which has little or no tendency to fuse. To line the channel with epithelium in order to create a real physiologic duct

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would hold out hope for more favorable and lasting results.

Since it is well known that corneal fistulas heal very poorly, if at all, it seems desirable to take advantage of this fact and to make the drainage opening in the peripheral zone of the cornea. However, the fluid from the anterior chamber should not flow directly into the conjunctival sac. It should first find its way into a thin-walled, epithelium-lined channel or duct. This duct should then open into the conjunctival sac at some distance. A sort of safety valve would thus be created, and aqueous fluid would force its way out into and through the drainage channel only during periods of increased intraocular pressure. At times of normal, or subnormal, intraocular pressure, the epithelium-lined channel would collapse and thus constitute an effective barrier

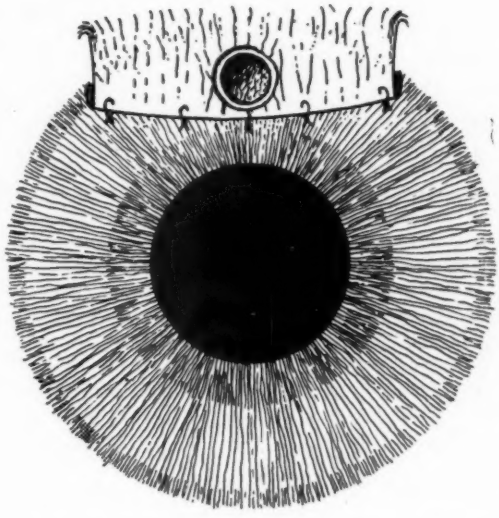

Fig. 3 (Lavine and Langenstrass). Sutures completed. Corneal trephine opening visible through second conjunctival flap.

against possible infection of the anterior chamber and also against the development of prolonged hypotonia.

The following procedure is therefore recommended. A very narrow strip of cornea is deprived of both epithelial and subepithelial tissue. The resulting corneal groove is arranged along a horizontal line under the upper lid. Five millimeters above the superior limbus, a conjunctival incision is made parallel to the corneal

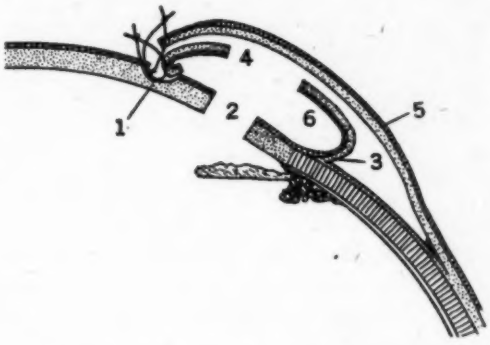

Fig. 4 (Lavine and Langenstrass). Diagram of result. (1) Corneal groove. (2) Corneal trephine opening. (3) First conjunctival flap, inverted. (4) Circular conjunctival excision. (5) Second conjunctival flap, not inverted. (6) Epithelium-lined epicorneal duct.

groove. A conjunctival flap is dissected free and reflected downward over the upper cornea. Its free conjunctival edge is sutured to the roughened area in such a way as to have its mesodermal surface rest against the raw tissue in the corneal groove. To cover the mesodermal surface of this conjunctival flap, a second conjunctival flap is brought down from the region of the upper fornix. Conjunctivocorneal sutures are drawn through its free edge and left untied. The next stage of the operation constitutes the creation of a corneal fistula. A small circular excision is made in the first, or central, conjunctival flap about 1 mm. inward from the limbus. A corneal trephine opening is now made through the center of that small conjunctival window. An Elliot trephine, 1.5 mm. in diameter, is used. A small piece of iris is excised beneath the region of the corneal fistula. The sutures of the second or outer conjunctival flap are now tied.

This operation creates a permanent

corneal fistula. That fistula is covered by an epithelium-lined conjunctival flap. This flap acts as a safety valve. The fistula is protected from infection by a fairly long epicorneal duct. That duct is entirely lined with epithelium. Corneal epithelium forms the floor. The conjunctival epithelium of the first, or central, conjunctival flap lines its roof. These two surfaces are smooth and have little or no tendency to fuse. This epithelium-lined epicorneal duct collapses (and thus guards the corneal fistula) at times of relatively normal intraocular pressure. The duct opens and functions as a capillary drain whenever the intraocular pressure exceeds a certain critical level.

Technical details of this procedure have been worked out in a series of operations on human cadavers and in animal experiments. We have recently had an opportunity to perform the operation on several patients with chronic glaucoma. We are not reporting clinical data in this preliminary communication, except to say that we were favorably impressed with the ease of execution of the method and with the smooth postoperative course. Whether the pressure reduction will continue to maintain itself indefinitely at a desirable level remains to be seen.

Summary

A theory is offered to account for the transient effect of many drainage operations in glaucomatous eyes. A new operation for lasting pressure reduction is proposed. A corneal fistula is connected with an artificially created epithelium-lined duct. The duct remains permanently open. It acts in the manner of a safety valve, closing the fistula at periods of normal or near normal pressure. The relatively long collapsible duct tends to reduce the opportunity for contamination of the anterior chamber to a minimum.

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A CASE OF FIXED STRABISMUS*

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The case which I am about to record is rare, and I have encountered no similar one in our national records and hardly any in the foreign literature. The patient is Mr. J. R. F., aged 60 years, residing at Mexicali, Lower California (Baja California). He came to see me on April 3, 1944. He was accompanied by a member of the family, because he could not obtain sufficiently useful vision even with the compensatory inclination of the head which is habitual in some cases of strabismus.

Inspection showed bilateral convergent strabismus, the most pronounced that I had ever seen in my professional practice. The cornea of the right eye was almost completely concealed at the internal canthus, and that of the left eye was exposed only as regards its outer half (fig. 1). The onset of the trouble had occurred 30 years previously, and there had been slow and progressive increase of the deformity to the point of complete incapacity for work. The patient's occupation had been that of a farmer.

No ocular movement could be demonstrated. There was a chemotic spot in the outer part of the left bulbar conjunctiva. There was apparently no action of the superior or inferior rectus of either eye, or of the obliques. It was impossible to investigate White's sign as to differentiation between involvement of the rectus and oblique muscles. After the usual laboratory examination, I proceeded to operation, in the performance of which I had the advantage of the assistance of Dr. Enrique Avalos González. The operation

* Read before the Asociación para Evitar la Ceguera en México, August 11 to 16, 1947.

† Translated by Dr. William H. Crisp, Denver, Colorado.

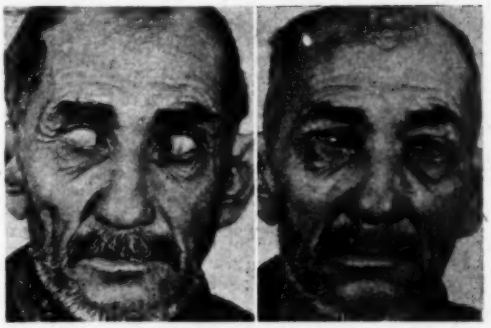
was performed on April 5th, beginning with the left eye. The plan was to execute retro-implantation of the internal rectus and the Blascowicz operation on the external rectus.

The conjunctival incision had to be made more or less blindly, because it was impossible to move the eye with the fixation forceps. I succeeded in taking hold of the internal rectus, and cautiously introduced the strabismus hook, using cautiously sustained but energetic traction.

I sectioned the insertion of the muscle. This behaved like a fibrous, inextensible, extremely short and tense band which fixed the eyeball in the nasal portion. Immediately upon division of this band, it was possible to move the eyeball in all directions. We proceeded to suture the internal rectus in front of the equator, in view of the possibility of subsequent overaction of the external rectus. We then did the Blascowicz operation on the external rectus, resecting this muscle to the extent of leaving the position of the eye somewhat overcorrected. The external rectus gave the impression of being narrowed and slender, and extremely flaccid.

At the moment of freeing the eye, and in the presence of decided limitation of the operative field, we failed to become conscious of adhesion of the inner and upper part of the cornea to the inner end of the lower eyelid in the region of the lacrimal punctum. There was thus exposed an adherent leukoma, and the manipulation resulted in perforation of Descemet's membrane, emptying of the anterior chamber, and an iris hernia. The last was excised.

Operation on the second eye was attended by the same difficulties as in the left eye. In performing the Blascowicz operation on the external rectus we were surprised to find a torn condition of a good many of the fibers of the muscle. This condition was explained when some days later an excellent colleague told me that he had attempted and achieved mobilization of this eye by means of fixation forceps!



Figs. 1 and 2 (Martínez). Showing the condition of the patient before and after operation.

The recto-implantation of the right external rectus was supplemented by two long fixation sutures which were tied firmly about 1.5 cm. beyond the external canthus, over a cushion of gauze, in order to insure overcorrection.

The postoperative course was extremely satisfactory, as may be noticed from Figure 2. The patient was dismissed on May 2, 1944, with perception of shadows for the right eye and vision of 5/10 in the left eye, without correction.

Discussion

There can be no doubt that the retraction syndrome resulted from fibrous degeneration of the internus muscles. In such a case it is sometimes impossible to establish the diagnosis completely before operation. After such an operation, normal movements are not to be expected, but from the esthetic and utilitarian point of view, as in the present case, the result is encouraging. Microscopic examination of the contracted muscle clearly shows fibrosis to be responsible for the strabismic fixation, although the essential cause for such a condition is not known. Fixation may be either unilateral or bilateral.

119 Avenida Pedro Loza.

SUBRETINAL HEMORRHAGE SIMULATING SARCOMA OF THE CHOROID*

A CLINICAL PATHOLOGIC REPORT

JOSEPH LAVAL, M.D.
New York

HISTORY

Mrs. G. M., aged 48 years, was seen by Dr. Benjamin Esterman on April 13, 1945, with a history of blurring and tearing of the left eye of three days' duration. The eyes had been entirely normal in the past and aside from having required reading glasses, they needed no attention.

Examination disclosed the right eye to be normal, with 20/30 vision corrected to 20/20, clear media, normal fundus and tension. The left eye had vision of 20/200 due to a large central scotoma extending downward and nasally. Inspection through a dilated pupil disclosed a pigmented mass at the macula, about 6 disc diameters in size and about 5 diopters elevated. It was quite round and extended to the upper temporal portion of the left disc. A retinal artery and vein coursed over its surface but none of the vessels in the fundus were engorged. There were no exudates, hemorrhages, or other pathologic conditions in any portion of the fundus. Ocular tension of the left eye was 16 mm. Hg (Schiøtz). There was no pulsation, exophthalmos, or limitation of motion of either globe.

A diagnosis of melanosarcoma of the left choroid was made and confirmed by Dr. R. T. Paton. A general physical examination disclosed no evidence of metastasis. The blood pressure was 120 mm. Hg (systolic) and 80 mm. Hg (diastolic). An enucleation was performed April 18, 1945.

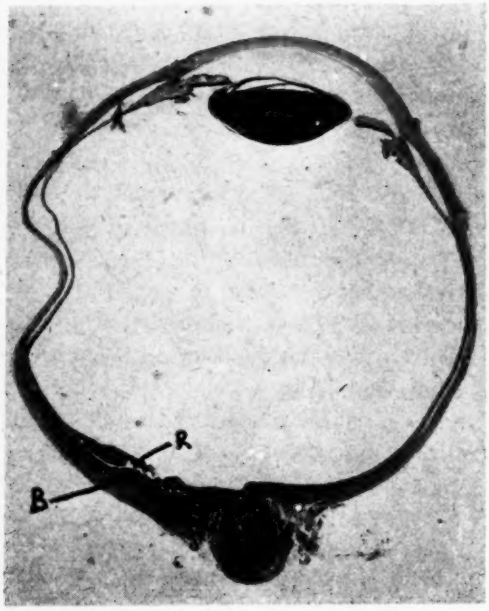


Fig. 1 (Laval). There is blood (B) lying between choroid and retina (R). The latter is detached as far as the disc in the region of the hemorrhage.

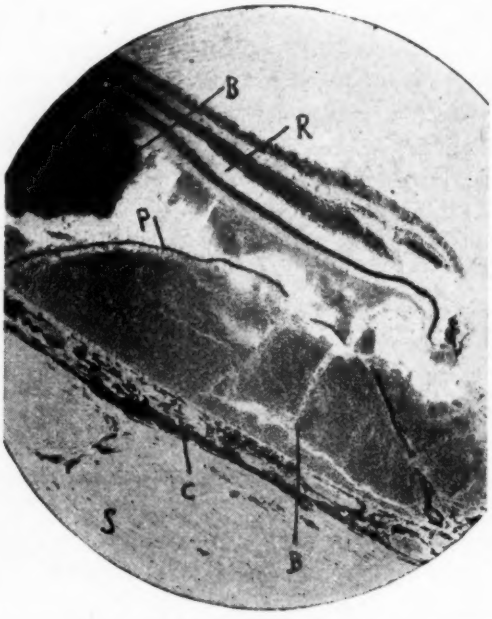


Fig. 2 (Laval). The pigment epithelium (P) is broken in three places allowing the blood (B) from the choroid (C) to break through and detach the retina (R). (S) is the sclera.

* From the laboratory of the Manhattan Eye, Ear and Throat Hospital.

PATHOLOGIC REPORT

The globe was normal in size and shape. There were no extraocular masses. On section, a dark solid mass was noted in the macular area extending to the disc (fig. 1).

Microscopic studies showed no abnormalities in the globe except for the region of the macula and the adjacent area (fig. 2). Here a large hemorrhagic mass was present, lying between the choroid and the detached retina. The pigment epithelium had been separated from the choroid by the mass of blood which had broken through the pigment epithelium in three distinct places. The blood which seeped through these three areas caused the detachment of the retina to follow. There was no blood in the vitreous. The choroidal vessels were quite full, but there was no evidence of sclerosis in the retinal or choroidal vessels. There was some perivascular infiltration with round cells at some of the larger choroidal vessels but no involvement of the vessel walls. Serial sections of the globe showed no evidence of tumor.

COMMENT

It occasionally happens that an eye is enucleated for a possible malignant melanoma only to find a subretinal hemorrhage on sectioning the eye. In the past 15 years this is the third time I have seen it at the laboratory of the Manhattan Eye, Ear, and Throat Hospital. This particular case is quite interesting because of the possibility of a disciform macular degeneration developing if the eye had not been enucleated. However, it is now two years since the original macular hemorrhage in the left eye and nothing has developed in the right eye. The retinal vessels in the right eye, according to Dr. Esterman who sees the patient a few times a year, are normal. The patient still has a normal blood pressure, her blood sugar is normal, and the urinalysis has never shown any abnormal findings. The perivascular infiltration of round cells about some of the larger choroidal vessels is the only indication of vascular disease and its significance is hard to evaluate.

136 East 64th Street (21).

BOECK'S SARCOID

REPORT OF CASE WITH AN UNUSUAL PRECIPITATING FACTOR

MORRIS KAPLAN, M.D.
Denver, Colorado

Boeck's sarcoid is generally considered to be a rather rare disease, and yet a perusal of recent literature proves it to be surprisingly common. The disease has been well described, especially by Levitt in the *Archives of Ophthalmology*, in 1941.

The eye is one of the favorite sites for this general disease and many cases are first seen by the oculist. In reports totaling 100 cases, 43 presented ocular involvement. Of these 43 cases, 28 were of uveitis, 7 involved the lids, 9 the lacrimal glands, and 6 the conjunctiva. Ten cases ended in phthisis bulbi and 7 of these 10 cases were bilateral. Thus seven percent of the cases ended in complete blindness. As is well known, the disease is almost identical with tuberculosis, differing from it only in absence of caseation in its tubercules and in absence of acid-fast organisms. The diagnosis is usually missed at first and is later established by tissue studies of biopsy specimens or by X-ray studies of the chest or of the phalanges.

CASE REPORT

The case herein described presents several unusual features. A 52-year-old woman, touring in South America, reached down to pet a dog in a private home. With great force, the dog bit her over the right eye. The upper jaw struck

the upper lid just below the brow and the lower jaw struck the lower lid at the orbital margin. The two resultant skin lacerations were treated almost immediately by a physician, who gave a prophylactic injection, the nature of which the patient did not ascertain. The eye itself seemed entirely uninjured. Three days later she noticed a painless swelling in the middle of the upper lid. This continued to enlarge for several days, causing a very noticeable bulging of the upper lid. It remained painless. About five weeks later, she presented herself at this office for treatment.

Just before she left for South America, she had had a routine refraction and examination at which time no appreciable irregularities were noted. She now presented this painless mass within or behind the right upper lid. It was freely moveable and measured 2 by 2 by 3 cm. The lid was quite protuberant and in mild mechanical ptosis. No other deviation from the normal was seen and the eye itself remained quite normal.

The mass felt exactly like an organized hematoma, which was considered as the most likely diagnosis. Local treatment consisted mostly of hot applications. Under this regimen, the mass became somewhat larger and moved from the midline of the lid to the outer third. The new ptosis was unsightly and, since the diagnosis now seemed obscure, surgical removal was decided upon.

Under local anesthesia the mass was easily isolated. It was found to extend back into the retrobulbar space into the orbital fat. It was easily removed. It was indurated, felt like cartilage, and had a yellowish hue. It obviously was not a hematoma. The wound healed uneventfully in a few days and the area resumed its normal appearance.

Laboratory report

Gross. The specimen consists of an elongated, gray-white to gray-yellow fragment of tissue removed from the right orbit. The sectioned surface is grayish white streaked with yellow.

Microscopic. Microscopic examination of the nodule removed from the region of the right eye shows it to be composed of fibrous tissue in which are well-circumscribed nodules consisting of epithelioid cells. In these collections are multinucleated giant cells of the Langhans' type. Cuffing the collections of nodules in some areas are small accumulations of lymphocytes. Caseation necrosis is not demonstrable. Special stains fail to reveal the presence of acid-fast bacilli.

Diagnosis. Boeck's sarcoid of the right orbit.

Internist's report

The patient was referred to her internist, who also had examined her thoroughly before she left for South America and had found no irregularities. She again underwent exhaustive study, and no appreciable abnormalities were found. X-ray reports were as follows.

Chest, both feet, and hands. Examination of this individual's chest reveals minimal spondylitis in the dorsal spine. There is no other evident abnormality. The dome of the left diaphragm is rather hazy and indistinct. The pleura is otherwise negative. The cardiac and aortic shadows are of normal size, shape, and position. The left hilar shadow is rather prominent, with a rounded configuration suggesting mediastinal adenopathy. The superior portion of the right hilar shadow assumes a rounded, smooth-bordered appearance which would be consistent with a small node on the right. The markings radiating from the left hilum into the left base are definitely intensified and more soft and granular than usually seen. The lung fields are otherwise essentially clear.

Summary. The findings in the chest are

not conclusive but may be considered supporting evidence of the pathologic report of biopsy.

Comment

Because of the biopsy report, anteroposterior views of the small bones of the hands and feet were obtained. There are small rounded, sharp, sharply demarcated excavations on the outer aspect of the articular surface of the terminal phalanx of the middle finger bilaterally and on the outer aspect of the right index finger. There are small osteophytes lying intimate with the joint spacing of the distal interphalangeal articulation of the middle fingers bilaterally. The excavations described could be on the basis of sarcoid depositions. The vertical trabecular pattern in the phalanges of the hands may be slightly exaggerated. This has been described as an early change in the presence of sarcoid. There is no other X-ray evidence of bone or joint change.

The internist recommended that no treatment be instituted and that the patient be observed at regular intervals.

It seems reasonable enough to agree with the pathologist that this is a case of sarcoidosis. It does not seem reasonable to assume that this sarcoid was caused by the dog bite, or could have been caused by any trauma, or could have grown so quickly if it had. It is not impossible that the tumor could have existed in the retrobulbar tissues above the eyeball and that the violent trauma could have caused its rupture forward behind the lid.

If a tumor of such size had existed two weeks before the accident when a routine examination was made, some interference with ocular movement should have been evident. This was not the case. There was no diplopia and no exophthalmos.

807 Republic Building (2).

THE PROBLEM OF STRABISMUS IN CHILDHOOD*

With especial reference to convergent strabismus in infancy and early childhood

James W. Smith, M.D.
New York

Superstition and fear on the part of misinformed parents, and the lack of a standardized diagnostic and therapeutic approach by ophthalmologists, are responsible in part for the unsolved problem of strabismus. Parents regard the eye as the most delicate organ in the body and view with horror the prospect of surgery on a child 2 or 3 years of age. They seek refuge in the story of the neighbor's boy who outgrew his esotropia —unaware that he only achieved this result after total and irreparable amblyopia. Usually a grandmother can augment the parents' fear with several examples of convergent strabismus followed postoperatively by divergent strabismus.

Blame for the latter result must be charged to ophthalmic confusion created by conflicting and often prejudiced schools of thought.[1] A convergent case can only be converted into divergence by excessive tenotomy of the internal recti muscles or failure to diagnose preoperatively the presence of a complicating vertical strabismus.

Causes of squint

Originally, I followed the teachings of Worth and Duane, but the neuro-anatomists and physiologists have challenged their views. Bielschowsky and Chavasse disagree in part with Worth and ignore

* From the Departments of Ophthalmology, New York Post Graduate Medical School and Hospital, Columbia University, and the Hospital for Joint Diseases. Read before the New York Society for Clinical Ophthalmology, January 6, 1947.

Duane's views on congenital superior rectus weakness. Chavasse[2] has originated a new entity—inhibitional palsy of the contralateral antagonist. Adler[3] upholds Bielschowsky's views that an isolated primary vertical motor paralysis is most commonly due to paralysis of the superior oblique muscle, but is willing to concede that the vast majority of cases of secondary vertical motor paralyses (that is, convergent squint associated with a noncomitant vertical deviation) more nearly fit into the picture of paralysis of a superior rectus.

I cannot understand the recent references in the literature regarding the frequency of superior oblique paralysis. Bielschowsky, in his large clinic at the University of Breslau, observed only 80 cases during the years 1923 to 1932. At a previous meeting in this Academy, I[4] reported only 50 cases from private practice over a 25-year period. When authorities disagree, the student, without facilities for guided clinical observation, must grope in the dark field of trial and error.

Conditions favoring development of squint

Conditions which favor the development of squint are anisometropia, opacities of the media, fundus disease, disorders acquired in infancy, and congenital anomalies that make the vision in one eye much worse than in the other. Other factors are heredity, hypermetropia, and the presence of a congenital vertical deviation. Chavasse considers "congenital defect of the fusion faculty" to be akin to a miracle and cites many traumatic and inflammatory lesions (prenatal and in infancy) that can produce muscle imbalance and acquired fusional impairment.

We operate too late to regain fusion and vision. Our so-called good results refer in the main to eyes that are cosmetically acceptable to parents. The child's disappointment is not evident until adult life when he is handicapped by the lack of binocular single vision and stereopsis.

Convergent strabismus

At what age does unilateral convergent strabismus manifest itself? In Worth's[5] series 13 percent of the cases appeared before the first year; 18 percent between 1 and 2 years; 25 percent between 2 and 3 years; 19 percent between 3 and 4 years; 11 percent between 4 and 5 years; 7 per cent between 5 and 6 years; and 7 percent after 6 years of age. In 75 percent of the cases, deviations were apparent before the end of the fourth year. Essentially, alternating strabismus appears even earlier in infancy. In 34 percent of the alternating cases, diagnosis was made before the first year, and in 19 percent, between the 1st and 2nd year of life. (13 percent were seen between the 2nd and 3rd year, and 16 percent between the 3rd and 4th year.) In 53 percent of the cases, the alternating strabismus was present before the end of the second year, and a total of 84 percent was apparent before the end of the fourth year. (About 2.3 percent of school children, aged 5 to 14 years, were found to have convergent strabismus, and about 0.2 of 1 percent had divergent strabismus.)

Constructive restoration

At what age does the infant manifest fusion? By the end of the first year the child evidences a definite desire for binocular single vision. We all know that the fusion faculty normally reaches its full development before the end of the sixth year. This fact, coupled with the high incidence of convergent strabismus before the end of the fourth year, indicates that our best opportunity for constructive restoration is between the ages of 2 to 6 years.

The exact measurement of the strabismus in degree of arc or prism diopters is

only of academic interest. Hirschberg's corneal light-reflex test, in which each millimeter of deviation represents about 15 prism diopters, is the best method that can be used with infants. After the second year more accurate determinations can be made with prisms and the cover test.

Estimation of the error of refraction is of vital importance. Under complete cycloplegia, retinoscopy reveals many cases with 2 to 8 diopters of hypermetropia and astigmatism. If the convergence excess is not complicated by vertical muscle imbalance, wearing glasses with full correction constantly will decrease the deviation or relieve the strabismus completely. In this age group, ointments induce more reliable cycloplegia than drops.

Visual tests for infants

A crude record of visual ability is desirable. By the time the average parent can be induced to bring an infant with fixed unilateral strabismus for examination, vision in the deviating eye is subnormal. My purpose in utilizing visual tests is to impress upon the mother how much damage has been done and to enlist her coöperation against further deterioration. The present-day tendency of waiting until the child can read the illiterate E or Snellen test types often reveals advanced amblyopia at the age of 3 or 4 years.

Evans[6] recently demonstrated his visual tests for infants, utilizing the movement of iron filings of various sizes on a white plastic tray. An infant interested in the filings being moved by a magnet on the underside of the tray will resent having its fixing eye covered thereby affording an excellent clue to amblyopia. At the ninth month the visual acuity is at least 6/72; at two years, 6/12; at three years, 6/9; and later, when the Snellen charts can be discerned, 6/6.[2] White marbles varying in size from ½ to 1½ inches in diameter can also be used to test vision in young children. This procedure can be performed in a few minutes and succeeds with most children old enough to walk. At a distance of 18 to 20 feet, the 1½-inch sphere corresponds roughly to 6/60 or 20/200 and the ¾-inch sphere, to 6/24 or 20/80. Improvement in vision after wearing glasses or following occlusion can be demonstrated to the parent as the patient proceeds to see smaller sized marbles.

Know child psychology

I have seen competent physicians who were long on knowledge but short on child psychology. Nervous mothers may cause children to cry in the waiting room, but the same children rarely display their temper in the presence of a sympathetic and kind doctor. The young child comes only for amusement. Its confidence, and your time, can be saved best by making a game out of the consultation.

Any device, musical or electrical, that engages the tot's attention should be utilized to gain your diagnostic goal. Come down to the child's level—even if you have to get down on the floor to observe muscle actions or for ophthalmoscopy or retinoscopy. Occasionally your best efforts will be thwarted by a spoiled brat. Be ready to admit failure cheerfully and send the bounder home with a lollipop or a penny. You will probably have better luck next time.

Muscle measurements in children are reliable only if the patients are completely relaxed. Unlike consultations on adults, time is unimportant. The child can return again and again until the necessary information has been obtained.

We see too many children with total amblyopia because some ophthalmologist found it inconvenient and time consuming to perform an accurate refraction upon or to treat an unruly child of 2 or 3 years.

The patients in the younger age range

afford us the best opportunity for correction of strabismus with less surgery, or in favorable cases without surgery. Even more gratifying is the preservation of binocular single vision by persistent treatment if started early.

Prescribing glasses

If the vision with glasses is subnormal in one eye, the ophthalmologist is duty bound to restore vision to normal, if possible, by atropinization or occlusion of the fixing eye. Early diagnosis and the early institution of treatment before amblyopia becomes marked will effect gratifying return of function in favorable cases within two months.

I have prescribed nonshatterable glasses in cases of accommodative convergent strabismus for patients 18 months of age. To the mother's amazement, the decrease in deviation and/or the visual improvement was so marked that the tiny tots insisted upon wearing the glasses constantly. These children will also permit the introduction of ½-percent atropine ointment in the fixing eye.

Occlusion to be effective must be total and continuous for days or weeks at a time. While this treatment is heroic and resented by the patient, it permits a rapid evaluation of the possibilities for visual restoration and decrease in the angle of squint.

Orthoptic training

Selected children between the ages of 3 and 6 years will coöperate with the orthoptic technician in an attempt to regain binocular single vision with the aid of one of the major amblyoscopes. Although the ideal time for fusion training is supposed to be prior to the sixth year, I have seen adults regain vision in an amblyopic eye following injury or disease in the normal eye.

When the strabismus is unilateral and these medical techniques are ineffective, early operation is indicated. Even the ardent advocates of orthoptic training deplore delay in these cases. I operate on high degrees of convergent strabismus with low insignificant hypermetropia between the age of 2 to 4 years. Only in these patients have I been successful in restoring binocular single vision.

Comment

Before concluding this topic, I want to stress the importance of diagnosing and treating convergent strabismus at the earliest age possible, if we wish to achieve normal vision in both eyes, binocular single vision, and normal fusion power. The therapeutic measures that may be employed to achieve an ideal result are: (1) Correction of errors of refraction. (2) Occlusion of the fixing eye. (3) Atropinization of the fixing eye. (4) Orthoptic training. (5) Surgery.

"Both as regards incidence of onset, and hope of cure, the age of innocence is the critical age."[2]

Divergent strabismus

Accommodative convergence insufficiency and nonaccommodative convergence insufficiency resulting from psychic disorders and debilitating systemic conditions are the chief causes of divergent strabismus in childhood. The early clinical signs are marked exophoria for near with normal near point of convergence. Later, in the stage of transient exotropia, the convergence near point recedes, exophoria is present for distance, and the prism divergence increases. If untreated, the transient exotropia is converted into a continuous strabismus. In the last phase, contracture of the external rectus and stretching and weakening of the internal rectus produces absolute impairment of inward and excess of outward rotation.

Examine for myopia

The arrest of these changes in the accommodative cases can be effected by a more vigorous approach to the problem of myopia. Children should be examined in the preschool period for stigmata of myopia. An exophoria for near or mild precrescent changes temporal to the optic nerve are premonitory signs of myopia, even in the presence of 20/20 and emmetropia. Too many children are permitted to indulge in all forms of convergence abuse, such as reading on their backs or even worse, sprawled on the floor with their eyes about four inches from their stacks of poorly-printed comics. When they are eventually referred by their school teachers, 3 to 5 diopters of myopia and varying degrees of transient exotropia are present. Early and full correction has forestalled the development of accommodative convergence insufficiency in about 10 percent of my cases with progressive myopia. Ocular hygiene should be explained to the parent and patient. Glasses must be worn constantly. The time interval between examinations must be set by the refractionist and not by the parent to avoid the necessity of marked increases in the lens prescription.

Fortunately, the fusion apparatus is fully developed in these patients and, after glasses have been prescribed, convergence exercises will do much to offset the advance of the divergence tendency.

Early management essential

Although we advocate early operations in convergent strabismus with low hypermetropia, in divergent strabismus, surgery is reserved for the advanced cases of long duration with extreme deformity. In fact, many cases operated upon prior to the age of 10 years will have a recurrence of almost the same degree of divergence requiring secondary surgery. A final word regarding congenital weakness of the elevator and depressor muscles. Many cases of divergent strabismus in which surgery not only failed but actually resulted in convergent strabismus are due to a complicating vertical muscle imbalance. In a previous paper,[7] I stressed the diagnostic importance of head tilt and associated skeletal deformities as positive evidence of vertical muscle imbalance in every case of convergent and divergent strabismus.

Once again, I would like to appeal for the early management of convergent strabismus. More patience with these little patients means perfect binocular single vision. Some of the divergence cases can be prevented or arrested by better ophthalmic supervision in the treatment of myopia.

1016 Fifth Avenue (28).

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SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

March 20, 1947

DR. BURTON CHANCE, M.D., *chairman*

ABSORPTION FROM THE VITREOUS

DR. LUDWIG VON SALLMANN (by invitation) presented a paper on "Some Factors Operative in Absorption from the Vitreous," an abstract of which follows.

In view of the difficulties in examining the fluid exchange in the posterior segment of the eye by the orthodox method, a new approach was selected with the practical aspect of the problem in mind. The experiments were grouped: (1) According to phenomena of diffusion in the vitreous and of its liquefaction. (2) In relation to the activity of the ciliary epithelium. (3) According to the influence of the basal metabolism. (4) On the basis of clinical methods used to speed up absorption from the vitreous.

The dependence of the diffusion of hemoglobin on the structural integrity and the viscosity of the vitreous was studied by injecting pigs' hemoglobin into fresh intact pigs' vitreous. In exposing the latter to a temperature of 37.5°C., the hemoglobin diffused throughout the vitreous within 18 hours; whereas, the spreading was very slow at refrigerator temperature. When hemoglobin was injected after autolysis of the vitreous had taken place upon storage of the preparations for 24 hours at room temperature, no difference in the diffusion of the hemoglobin was noticed at various temperatures. It was concluded that autolytic liquefaction, or lowering of the viscosity of the vitreous, had resulted in the speedy diffusion in eyes which had been exposed to 37.5°C. in the first series of experiments.

The effect of liquefaction on diffusion or spreading and absorption of foreign matter was investigated in an extensive series of *in vivo* experiments on 24 rabbits. Partial liquefaction was induced by injecting various amounts of a purified and concentrated hyaluronidase prepared by Dr. Karl Meyer from bull testes; this mucolytic enzyme acts on the substrate of the vitreous. The enzyme dilutions were introduced together with suspensions of the animals' own red cells, which served as the test substance. Early spreading of the red cells and moderately accelerated absorption were observed clinically and histologically in the eyes which had received more than 10 gammas of the enzyme. When the dose exceeded 50 gammas, the treatment elicited a conspicuous inflammatory response from the inner membranes of the eye. Even 2 of 5 eyes of the 50-gamma series reacted with marked signs of inflammation. The rest of that group and the eyes injected with 10 or 25 gammas exhibited early loosening of the clot of red cells and somewhat earlier absorption without apparent toxic side action. It seemed that the partial liquefaction induced by the mucolytic enzyme caused the early spreading of the red cells in the vitreous, and this in turn may have facilitated the speedier breakdown of the erythrocytes and their absorption. It is evident that at the present time the toxic action of a medium dose of the enzyme obviates its practical use.

The problem of active absorption possibly exercised by the ciliary epithelium was studied on the basis of R. Hoeber's

work on the active transfer of a group of sulfonic dyestuffs through the wall of the proximal tubules of frogs' kidneys. Hoeber found that this transfer depended on the polar-nonpolar configuration of the dyestuffs, since the dyes, with the sulfonic groups attached to one of the benzene or naphthalene rings, were transferred through the wall in contrast to the dyes in which each of the benzene or naphthalene rings carried a sulfonic radical. The present experiment on albino rabbits, albino rats, and frogs, with the use of various techniques, did not give evidence that the dye in the vitreous had penetrated the ciliary cells or had accumulated in intracellular vacuoles except in one instance among 60 eyes. Despite the negative results, further studies are warranted.

The influence of the basal metabolism on resorption was again studied on suspensions of the animals' own red cells injected intravitreally. Five litters of albino rats (34 animals) were divided into three groups so that litter mates were distributed among the three series. The first group of 12 rats were thyroidectomized by Dr. G. K. Smelser when the rats were two weeks old. The second group of 12 animals received daily subcutaneous injections of thyroxin (1 mg. per 100 gm. body weight). After the injection of a small amount of the animal's own red cells, the density and extent of the cell deposits in the vitreous were estimated by ophthalmoscopy and graded. The 68 eyes were examined three times a week, and the graded findings were tabulated so that the resorption could be expressed in curves when plotted against time. Thyroxin had no clear effect on the growth of the animals or on resorption of red cells from the vitreous when compared with a control series of 10 untreated normal animals. The thyroidectomized rats showed greatly retarded growth and, in general, flat resorption curves; that is, the red cells disappeared more slowly from the vitreous than in the 20 control eyes.

The last group of experiments was designed along the lines of clinical methods used in the therapy of hemorrhages and other opacities of the vitreous. The effects of subcutaneous and subconjunctival injections of hypertonic salt solutions, of iontophoresis, and of artificial fever were examined. The animals' own red cells or a concentrated solution of inulin introduced into the vitreous was the test substance. Only the short bouts of fever repeated daily for two weeks had, in rats, an indisputably beneficial effect on the rate of disappearance of the red cells from the vitreous (40 animals).

Discussion. Dr. Irving H. Leopold. The vitreous humor has been the subject of investigation by numerous men for many years and has involved a knowledge of special techniques, of physical chemistry, and of ophthalmology. Duke-Elder, Robertson, Hodgeson, Goedbled, Baurmann, and others have worked on this problem for years. None have contributed more to our present knowledge of the vitreous than has Dr. von Sallmann. It was a pleasure to hear him speak on aspects of the vitreous humor, because on this subject he is certainly an authority.

The vitreous humor is a colloidal structure, and as a colloidal structure may exist as a sol or a gel. Sols are colloidal states in which the solid particles are dispersed in a fluid medium, and gels are colloidal states in which the solid particles, so to speak, join hands, and form a framework in which the liquid medium is dispersed.

Most of the evidence, and much of the more recent evidence has been contributed by Dr. von Sallmann, is in favor of the vitreous humor being a gel. It is not a very stable gel. The liquid and the solid phase are in an unstable equilibrium, and

this can be upset easily. When the vitreous gel is disturbed, the transparency of the vitreous is altered.

There are two ways, roughly, in which the vitreous transparency can be changed with the production of vitreous opacities. First, by disturbing the gel of the vitreous and secondly, by the influx of new materials such as red cells, exudates, and foreign bodies. Examples of the first mechanism are senile vitreous opacities. These are thought to be due to the aging of the gel, changing it into the sol state. All gels tend to break down to the sol state as they age. Myopic vitreous opacities are thought to result from movement of the vitreous gel in an enlarged globe. Liquefaction of the vitreous gel by proteolytic enzymes may result in opacities.

The other group of opacities are due to the influx of new material, such as red cells, which Dr. von Sallmann discussed tonight. We all recognize the problem of intraocular hemorrhage as a serious one. Anterior-chamber hemorrhage is still an unanswered therapeutic problem, but vitreous hemorrhage offers an even greater therapeutic problem. In vitreous hemorrhage we are not only concerned with the resorption of blood, but we have to maintain the status quo of the vitreous gel; or reconstruct it, if it is disturbed. Dr. von Sallmann has given us some ideas on the method of reabsorption of the cells, but at the same time has suggested methods which disturb the structure of the vitreous body.

It is interesting to observe workers in other fields of medical science turning to the eye as an aid in solving their own problems. This has happened in the case of the spreading factor. Dr. von Sallmann has reviewed their observations and added more of his own in order to solve some ophthalmologic problems. Duran Reynals found that if vaccinia virus were injected into the testes of rabbits, one could be more certain of takes than if the injections were made in other areas of the body. He could get much more severe infection by such injections. He then found that if an extract of the testes were injected into the skin prior to vaccinia injections, he could also facilitate systemic spread of the virus and increase the number of takes. He thought that the testes extract facilitated the spread of the vaccinia by hydrolyzing the viscous mucoproteins in the connective tissues.

These spreading factors were then found to hydrolyze hyaluronic acid. As you know, Karl Meyer and his coworkers isolated this mucopolysaccharide from the vitreous humor. Other investigators then turned to the vitreous humor, an excellent substrate of hyaluronic acid, as a place in which to compare and study the activity of various spreading factors. They measured the ability of the various spreading factors to reduce the viscosity of the vitreous humor. Most spreading factors are thought to act through hydrolysis of hyaluronic acid. However, ascorbic acid is also known to reduce the viscosity of the vitreous. It does not work theoretically, as the other spreading factors, and for that reason, many people do not consider it a spreading factor. Nevertheless, it can, when properly employed, reduce the viscosity of the vitreous humor, and it is quite possible that it may help in this manner in the resorption of vitreous hemorrhage. Vitamin C has been used in the past as a therapeutic agent in intraocular hemorrhage. It was thought that it built up intercellular cement substance and reduced the capillary permeability, thus preventing further bleeding. It was not generally realized that it might reduce the viscosity of the vitreous, and facilitate greater distribution of red cells and earlier resorption. It may not be as toxic as hyaluronidase and, perhaps, can be injected directly into the vitreous without

producing the untoward effects of hyaluronidase.

The observations of the influence of thyroidectomy on reabsorption of blood are worthy of note. In 1936, Jeandelize and Drouet reported on cases of recurring vitreous hemorrhage. They reported that all of their patients with this disorder had some degree of hyperthyroidism, and felt that they could prevent recurrences by irradiating the pituitary body thus producing lowered thyroid activity. It is interesting that the lower thyroid activity may work toward the prevention of hemorrhagic recurrences, and at the same time retard the resorption of existing hemorrhages.

For years fever therapy or shock therapy have been recommended for resorption of vitreous hemorrhage, usually without any well-founded, controlled experiments to back up the recommendations. Dr. von Sallmann has given us controlled data to show the value of artificial fever.

There is some question about the circulation of fluid in the vitreous humor. As you know, the vitreous body is not thought to have any metabolism. It is supposed to be a product of the surrounding cells. It probably does not utilize oxygen or produce carbon dioxide. Regardless of this lack of necessity for metabolic interchange, there is some evidence of a flow of fluid through the vitreous. This has been demonstrated in rabbits, but not satisfactorily in man. This flow is thought to be from the ciliary body toward the optic nerve. Certainly the diffusion of the red cells would not tend to substantiate the idea of such a direction of flow in the vitreous. The diffusion was in all directions from the site of injection into the vitreous.

Dr. Francis Heed Adler. I had not intended to discuss this paper largely because of a feeling of inadequacy. I rise, as they say in parliamentary language, to a point of order; that is, to object to the statement in Dr. von Sallmann's speech in which he mentioned my name as contributing to our knowledge of the vitreous. Although I have been interested in some of the physiologic problems of the vitreous, the work to which he referred is chiefly work in my laboratory done by Dr. Irving Leopold, and the full credit should be given to him.

It seems to me from what has been said about hyaluronidase that that is practically a closed door. The danger threshold between the effect that is experimentally demonstrable lies too close to production of retinal damage, which is also demonstrable. This is comparable to some anesthetics, which perhaps are good anesthetics, but the threshold of anesthesia is very close to that of death.

I would be interested in having Dr. von Sallmann comment further on the question of thyroxin, particularly in relation to the water-binding properties of thyroxin, and on the anterior pituitary hormone and the part it plays, in producing exophthalmos. I wonder if he has taken into consideration the relation between thyroxin and the anterior pituitary hormone in this particular condition, and whether he has anything further to say on this particular subject, because I think that it is a lead which offers us something.

Finally, I can confirm from the clinical point of view the effects of fever therapy on the absorption of vitreous hemorrhage. It is certainly one of the chief therapeutic measures that we have to draw on at the present time.

Dr. Ludwig von Sallmann. I want to thank Dr. Leopold and Dr. Adler for their interesting discussion. I am sorry that I cannot offer anything on the relationship of the thyroxin experiments to the pituitary function. I did not go into detail on the thyroxin injections, because the results were rather erratic. Sometimes

a fairly significant increase in absorption of the red cells was obtained, but in other instances there were flat absorption curves.

Dr. Francis Heed Adler. Why did you use thyroxin? What theoretical consideration led you to suspect that you might get some effect from thyroxin on the absorption of the vitreous hemorrhages?

Dr. Ludwig von Sallmann (closing). Hans Eppinger studied the effect of thyroid preparations on the water threshold, in 1914, and he described the speedy absorption of tissue fluid in certain types of edema. These observations on the water threshold and the stimulation of the basal metabolism by thyroxin were the theoretical bases of my experiments.

George F. J. Kelly,
Clerk.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

January 6, 1947

DR. BENJAMIN FRIEDMAN, *president*

The program was devoted to a discussion of ophthalmic problems in children.

VISUAL FUNCTIONS IN INFANTS

DR. ALFRED KESTENBAUM said that the time of development of the various forms of eye movements were shown in tests performed on about 70 infants. Optically elicited movement (O.E.M.) means movement toward a peripherally appearing object and hence is a function of a peripheral retinal point. The O.E.M. is a gliding movement which tries to keep the image of a moving object on the macula and hence is a function of the macula. Up to the age of 4 to 6 months there is no gliding follow movement; it is substituted by a cogwheel movement. Optokinetic nystagmus appeared at the age of 3 to 6 months. Acoustically elicited eye movement appears very late, usually not before six months of age. Vestibular reaction of the eye on head rotation, present immediately after birth, was found to be at first in the form of a simple deviation.

Dr. Kestenbaum stated that visual acuity can be estimated by the following procedures: (1) Examination of the direct and of the indirect pupillary reaction: at birth; (2) Marcus Gunn's pupillary test (for optic-nerve lesions): at birth; (3) pseudo-anisocoria test (for optic-nerve lesions): at birth; (4) follows movement test (for disturbance of macular vision): 4 to 6 months; (5) optokinetic nystagmus test (for disturbance of macular vision): 3 to 6 months.

The field of vision can be examined by the O.E.M. test in the form of the fact-test at from 2 to 4 weeks. Eye motility can be studied by: (1) O.E.M. test (possible only if the visual field is preserved): 2 to 4 weeks; (2) vestibular compensation test: at birth. Phoria and tropia can be studied by screening tests.

Marcus Gunn's test consists of alternation between illumination of the right eye with occlusion of the left eye and illumination of the left eye with occlusion of the right eye. If in the first case a pupillary contraction occurs, but in the second a pupillary dilatation occurs, the presence of a lesion of the nerve fibers of the left eye may be assumed. This test is modified in the pseudo-anisocoria test in which the finally resulting pupillary sizes, after occlusion of the right eye, are observed and not the movements of the pupil. After occlusion of the left eye, the size of the pupils are exactly measured and compared with each other. The latter two signs are especially valuable in the diagnosis of retrobulbar lesions. Inability to follow a moving object and absence of optokinetic nystagmus indicate disturbance of macular vision. In the O.E.M. test, an object is brought into the field from without. When the eye turns toward the ob-

ject, it proves that the object has been seen. Systematic application of this O.E.M. test in eight directions gives a fairly good visual field. Dr. Kestenbaum concluded that the strongest attraction on the infant's eye is exercised by a human face entering the visual field.

Management of infantile glaucoma

Dr. David H. Webster reported the operative results in six cases of infantile or congenital glaucoma. All the cases were operated during the first week of life. Three cases in which the tension remained normal following iridencleisis did not develop buphthalmos. One case in which aniridia was an associated anomaly did not do very well following an attempted iridencleisis. In spite of repeated trephinations development of buphthalmos occurred in two cases.

In the course of 35 years, Dr. Webster said that he had seen eight cases. The condition is recognized by the steamy white cornea, 14 to 16 mm. in diameter. The creation of a new filtration channel to compensate for the structural defects in the angle of the anterior chamber is accomplished by iridencleisis.

Retrolental fibroplasia

Dr. Algernon B. Reese stated that for over a hundred years basically related lesions have been reported under various synonyms including tunica vasculosa lentis, congenital fibrous tissue back of the lens, congenital connective tissue in the vitreous with and without detachment of the retina, remains of the hyaloid system, and so forth. Formerly such lesions were rare, and occurred in full-term infants, and were usually unilateral. During the last 10 years there has been an increased incidence of these lesions which have been reported in premature infants and bilaterally.

Dr. Terry has called these more recent cases retrolental fibroplasia. He believes that the condition appeared after birth (not before the fourth month). He considered it a newly acquired disease.

Dr. Reese said that Dr. Payne and he believe that both lesions mentioned are one and the same, basically. He felt that the lesion is congenital, although at times the matrix may be inconspicuous at birth and becomes more pronounced after birth by hyperplasia. He explained the greater incidence on the basis of the fact that more premature children now survive. He called attention to the fact that this eye lesion is frequently associated with hemangiomatous manifestations elsewhere.

Dr. Reese cited the opinion of Dr. Krause that this lesion is characteristically associated with mental retardation, and that these two manifestations are due to a faulty development of the neuroectoderm over that portion where it forms the retina as well as the brain. Dr. Krause called the condition congenital encephalo-ophthalmic dsyplasia. Dr. Reese said that he did not agree with Dr. Krause's concept because:

1. The eye lesion is not characterized by a dysplasia of the retina but by the presence of fibrous tissue in the vitreous. This fibrous tissue may contain any of the derivatives of the mesoderm such as cartilage, smooth muscle, fat, connective tissue, and blood vessels.

2. Remains of the hyaloid artery, or some of this system, is a common finding and would not be expected in retinal dysplasia.

3. Mental retardation due to a brain dysplasia or agenesis does not seem to be an integral part of the lesion. Excluding the cases of hydrocephalus, only 30 percent of these cases show mental retardation. It seems more likely that the mental retardation present in these cases is due to prematurity (82 percent of Dr. Krause's cases were premature), to cerebral anoxia, and to hydrocephalus. Furthermore, autopsy findings presented by Dr. Krause did not seem convincing re-

garding the pathologic evidence of a cerebral agenesis.

Dr. Reese stated that the etiology of this eye lesion is not clear. It seems likely that the same factor producing the prematurity also produces the eye lesion. The mothers of these children give a high incidence of bleeding during pregnancy, and a history of respiratory infection is common. The affected eyes sometimes show evidence of inflammation. This is manifested by residua of iritis or choroiditis, and microscopically by the presence of round cells in some of the cases. There are instances of subclinical maternal infections producing congenital anomalies in the eyes of the offspring. Dr. Reese cited the case of a bilateral retrolental fibroplasia occurring in the offspring of a mother who had rubella in the fourth month of pregnancy.

Discussion. Dr. Kornzweig questioned the possible time of occurrence of the toxic influence. It must occur after full development of the hyaloid system. The hyaloid system reaches the height of its development during the second to third month, then it atrophies. The ciliary processes are formed after the third month and touch the lens after the fourth month. The toxic influence must, therefore occur between the third and fourth month concluded Dr. Kornzweig.

Dr. Sitcheska agreed that these patients have well-developed mentality.

Dr. Mandelbaum asked if it is always possible to see the vascular structure of the tissues or whether the lens is too dense.

Dr. Friedman asked whether X-ray studies show areas of calcification, as in glioma. He remarked that in these cases the pupil does not dilate well and if atropine is used glaucoma may result.

Dr. Reese (in closing) replied: (1) That of 18 patients, 16 were normal mentally. (2) In answer to Dr. Mandelbaum's question, that cataractous changes can occur and prevent the view of the tissues and also a cloudy cornea may prevent it. (3) In answer to Dr. Friedman, that X-ray studies do not show any calcification.

Congenital cataract after maternal rubella

Dr. Charles Perera stated that the occurence of congenital malformations (cataract, cardiac abnormalities, mental retardation, deaf-mutism, and other defects) following maternal rubella was reported from Australia in 1942 by Gregg, and confirmed the following year by Swan and his colleagues. Reese brought this to the attention of American ophthalmologists in 1944. Since then a number of reports have appeared on this subject.

The ocular pathologic changes reported by Swan in three infants dying from congenital defects, by Terry, and by Cordes and Barber in an 8-week-old embryo showed retardation in the development of the involved eyes, necrosis of the nuclear portion of the lens, and degenerative changes in the equatorial lens fibers. These findings are consistent with the clinical observations of microphthalmos, nuclear cataract, and diffuse lens opacification in these children. Microscope preparations of the eyes of a 10-week-old fetus, obtained from a woman who had rubella during the seventh week of pregnancy, show degenerative changes in the fetal lenses in the central and posterior portions.

A patient seen in November, 1946, was the 3-year-old daughter of a woman who had rubella late in the second month of pregnancy. The girl was unable to walk or talk, and had diffusely hazy lenses and a variable esotropia.

Dr. Perera discussed the treatment of congenital cataract with special reference to extraction of the lens, needling, and through-and-through discission of the lens.

This type of cataract may result from the passage of the virus or toxic substances through the placenta into the fetal circulation or through the amniotic fluid. He stressed the need for further investigation of this subject by studies in the field of pathology and by more surveys such as those reported from the Vanderbilt University Hospital, in 1945, by Conte, McCammon, and Christie.

Discussion. Dr. Smith asked whether discission should be performed during the first or second month of life, or be postponed to the 11th to 12th month. Dr. Smith also agreed on the use of a through-and-through discission because of the danger of glaucoma.

Dr. Perera (in closing) replied that in his opinion early operation is advisable before the sixth month, preferably during the second or third month.

STRABISMUS IN CHILDHOOD

DR. JAMES W. SMITH presented this subject. His paper is published in full on page 85 of this issue of the JOURNAL.

Bernard Kronenberg,
Secretary.

SOCIEDAD OFTALMOLOGICA DE MADRID

February 21, 1947

PROFUSE HEMORRHAGE IN PTERYGIUM

DR. MARIN AMAT and DR. MARIN ENCISO presented the case of a woman, aged 57 years, who was admitted to their service in the ophthamologic clinic of the Provincial Hospital in Madrid. The patient presented a chronic suppurating dacryocystitis and an internal membranous pterygium in both eyes. She was operated on and both lacrimal sacs were removed in one session without anything abnormal occurring either during the operation or after the operation.

A few days afterwards, on December 3, 1946, they removed in one session both pterygia similarly without the least complications. The postoperative course was perfectly normal and on the seventh day the bandage was removed. But during that night the right eye began to bleed and it was necessary to bandage the eye again in order to check the bleeding. The next morning the bandage was removed. On the night of the same day, another hemorrhage occurred in the small operative wound of the pterygium of the right eye, but this hemorrhage was very profuse. It was not possible to check the bleeding with a compression bandage because the latter became immediately saturated with blood. The doctor on duty had to renew the bandage several times and give many coagulating injections of cebion E (vitamine K), coaguleno, and zimema, without being able to stop the bleeding effectively.

The following morning the patient was in shock (cold, with a barely perceptible pulse, with vomiting), necessitating the giving of a subcutaneous injection of 500 cc. of physiologic saline and also a transfusion of 300 cc. of whole blood. By these means the hemorrhage was stopped and did not recur. Thereafter the patient slowly improved and was discharged as cured on December 23rd.

In view of such an important complication we undertook to investigate the etiology, but nothing in the family history or the biologic analysis gave any clear insight into the cause. The only outstanding factor was that the woman was in very poor circumstances and had subsisted for a long time on practically a hunger diet, which indicated that she was in a state of avitaminosis with a consequent lack of vitamin K and a reduction in the production of thrombokinase.

PSEUDOGLIOMA OF METASTATIC ORIGIN

This was a case of a little girl, aged 26 months, who showed a morbillous exanthem with temperature and sudden appearance. All of the symptoms disappeared the following day. The next day

she awoke with her whole body swollen. There was intense oliguria and an analysis of the urine showed albuminuria, hematuria, and cylindruria. Two days passed without any change in the picture. On the fourth day of the sickness there appeared a whitish spot, ring shaped, which covered a large part of the pupil of the right eye. A similar spot appeared on the left eye the following day. The eyes presented a picture of a bilateral uveitis with an intense ciliary reaction and with hypopyon. On the same day penicillin was administered (27,000 units every three hours) until a total of 1,700,000 units had been given. The general condition became improved. The spot in the left eye disappeared in the first 24 hours and that on the right eye after about 10 days.

When the child was examined on January 17th, there was a slight circumcorneal congestion and dilated pupils without reaction to light but without any inflammatory manifestations at the anterior pole. With the ophthalmoscope could be seen a yellowish white mass in the vitreous body in both eyes which prevented a view of the fundi of the eyes and verified the absence of vision. There was hypotension in the right eye.

Three phases in this clinical history stand out: (1) general symptoms; (2) the false picture of nephritis, which showed the time that intervened between the general infection and the ocular metastasis; (3) the presence of ocular symptoms.

The diagnosis is a pseudoglioma of both eyes of metastatic origin subsequent to a septic process from which the patient had suffered. Prognosis as to vision is unfavorable because one could not hope for any recovery of vision. The chief point to emphasize is the success of penicillin in the treatment of ocular metastasis without which, in this case, probably both eyes, certainly one of them, would have terminated with a panophthalmitis.

Foreign body in sclera

Dr. Marin Amat presented a case of a foreign body embedded in the external sheath of the sclera alongside the optic nerve after having crossed the eyeball. It was a case of an accident of occupational origin, relating to a railroad worker who, while working on the track on January 21, 1947, was struck in the right eye by a foreign body. Radiographs of the orbit were immediately taken, one frontal, one lateral, the latter in two exposures (while looking up and while looking down) which were positive and showed the presence of an intraocular foreign body.

Consequently, a search was made by the scleral or posterior route and the proper point of the giant electromagnet was introduced into the operative wound. Notwithstanding repeated trials, removal failed and enucleation of the eye became necessary. A couple of days afterwards the eye was removed. On dissecting it the following details were noted: (1) Total detachment of the retina with abundant yellowish subretinal liquid. (2) Absence of any intraocular foreign body. (3) The presence of a circumscribed hemorrhagic focus in the ciliary region and an extensive zone of whitish infiltration in the process of organization. (4) What had seemed to be foreign bodies in the lens and in the retina were nothing more than a true tattooing by the iron salts. (5) The hard nodule alongside the optic nerve showed on dissection to be a small piece of iron, triangular in shape with a pointed vertex, which was imbedded in the external sheaths of the sclera, in Tenon's space, and encased by reparative tissue arising from the external sheath of the optic nerve and from Tenon's capsule. These explained the various findings which resulted from the path of the foreign body, and also explained the false radiographic localization.

Joseph I. Pascal,
Translator.

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OPHTHALMIC PUBLICITY

At the recent meeting of the American Academy of Ophthalmology and Otolaryngology, there were two important contributions, the public and press reactions to which merit some comment. These two contributions both dealt with procedures which had been highly and luridly publicized in the lay press. Both contributions were largely negative in their conclusions. Through the public relations officer of the Academy, the representatives of the press were authoritatively informed of the negative results. These negative conclusions, the results of long and patient investigation, received, with one or two notable exceptions, little or no comment in any of the newspapers, whereas the gaudy claims of the proponents of these forms of therapy, claims based on personal enthusiasm and unsupported by scientific investigation, had previously been heralded far and wide across the country by the various news agencies.

The first of these contributions related to

the corneal-transplant operation. The careful studies of the five speakers in the symposium showed the operation to be one of limited applicability, of uncertain results, with many contraindications, and was especially contraindicated in patients with vision of 20/100 or better, where there was a greater chance of permanent blindness than of improvement of vision. At best, in the over-all statistics, only approximately one third of the patients undergoing this operation had material improvement of vision. It was quite true, however, that in specially selected cases, notably in keratoconus and slight central corneal scarring without undue vascularization, spectacular visual improvement, not possible by any other means, might be expected in a materially higher percentage of cases.

The second contribution dealt with the results of tissue therapy and the administration of cod-liver oil in patients with pigmentary degeneration of the retina, the so-called Filatov treatment. Here a careful investigation of the results of this treatment in a large number of patients failed to reveal any evidence supporting the wide claims made by the proponents of this form of therapy. A limited number of the patients showed some slight subjective improvement in the usual level, but in no greater number and of no higher degree than had been reported with the other widely different forms of treatment formerly proposed—vitamin therapy, sympathectomy, estrogenin and androgenin treatment, vasodilators, and reduction of the intraocular pressure. Objective examination of these patients, before and after treatment, showed little or no change in the visual fields or light adaptation. The conclusion appeared inescapable that, so far as this careful investigation was concerned, there was no evidence at hand to support the extravagant claims which had been so widely made.

At first glance it appears surprising that the various news agencies, whose function it is to disseminate "news," and the lay press of the country, which should strive to lay pertinent information before the public in readable form, exhibit little or no interest in negative results. On thought, however, the answer appears easy. Negative results are not "news," and the publication of results invalidating already published claims is, at best, only a wet blanket and, at the worst, may precipitate on the heads of the publishers recriminations and even damage suits.

A case in point which well illustrates this is the attitude recently taken by the Curtis Publishing Company. The question of various eye exercises for myopics had been widely publicized, and indeed apparently endorsed by a number of prominent periodicals. The Curtis Publishing Company had taken no part in this publicity, but nevertheless was deeply interested in its possible humanitarian application and its publicity value. Through the agency of the editor-in-chief of one of their major publications, they undertook to finance a thorough investigation of the possible beneficial results of such treatment. As a result of their offer, a joint investigation of this treatment was undertaken by a group of psychologists and optometrists on the one hand, and the team from the Wilmer Institute staff on the other. The Wilmer Institute team acted only as judges of any results observed. The results of this study, published in full in the medical press, were largely negative. These negative results were duly transmitted to the Curtis Publishing Company who, since they had financed the psychology-optometry phase of the investigation, had just right for first publication after the reports had appeared in the medical journals. However, in as much as the results were negative, the Curtis Publishing Company had no interest in publishing them or commenting upon them. They simply charged off their investment. The publication of the results, from their standpoint, might only have touched off a controversy, uninteresting to the great body of their readers, and in which they had no inclination to participate.

Such a specialty as ophthalmology is par-

ticularly susceptible to the ill results of misleading publicity. The blind have an emotional appeal to the seeing world. Any form of treatment which promises to alleviate or cure blindness is immediately of interest to the general public, any claims supporting such treatment find sympathetic ears, and any agency endorsing the proposed form of therapy finds ready supporters. In short such forms of treatment are interesting and appealing "news," which the public is anxious to read and the press, therefore, correspondingly anxious to publish. While, undoubtedly, in many instances the publicizing of these unproven forms of therapy is animated by the most altruistic motives, nevertheless in other instances this does not appear to be true. It must regretfully be admitted that there are a few ophthalmologists who avail themselves of this sentimental appeal for the purpose of self-advertising. Were the news agencies as industrious in circulating the results of investigations which disprove these spectacular forms of treatment, and the lay press as eager to print them as they are to publicize the original claims, it might well have a deterrent effect on the self-advertisers.

Although the reaction of the press to negative results is understandable, the question may well be asked—does it reflect a healthy interest in the public welfare and is it in line with the humanitarian motives which one would assume underlay the dissemination of information relating to these apparent cures? There the answer should be an unequivocal "No."

As was pointed out in the discussion of the paper on pigmentary degeneration of the retina, mankind is afflicted with a host of maladies, often congenital or degenerative, about which the conscientious physician can do little or nothing. These unfortunates are constantly seeking for some relief or cure of these conditions for which they can obtain little alleviation from legitimate doctors. They are ripe fruit, ready for the plucking. As the late Sir William Osler once remarked, "People love to be fooled, and the sons and daughters of Circe are still in our midst." When there is a demand, there is usually a supply. The publicization of unproven and often utterly fallacious remedies is followed by a deluge of inquiries on the doctors and medical centers for information as to their value. There must then ensue a long period of patient investigation to validate or invalidate these claims. If the claims are validated, as is rarely the case, the fundamental investigator receives but scant public recognition—the bulk of the glory goes to the proposer or instigator of the treatment, to the man who thought up the dream. If the claims are invalidated, the investigator gets no thanks for his labors, and may often be the subject of attack in one form or another. At the best the whole incident is quickly forgotten.

The lay press cannot escape its responsibility. If the press is willing to publish the unfounded and unproven claims of enthusiasts and self-advertisers, to give them wide publicity, and often to make them the subject of special articles and Sunday features, it should be equally willing, even anxious, to follow up the subject and give equal publicity to legitimate releases of investigators which disprove the former extravagant claims. If false hopes are aroused in the minds of sufferers from untractable ailments by injudicious publicity, the same agents responsible for the publicity should do what they can to rectify their error. Only thus can their public duty be discharged. The present policy of exploiting sensational claims and soft-pedaling the results of investigations which disprove these claims, is playing into the hands of self-advertisers and, in some cases, of charlatans.

Alan C. Woods.

ECLIPSE RETINITIS

On November 12th there occurred a partial eclipse throughout the greater part of the United States. Along the Eastern Sea-

board the time of its visibility varied between 16 and 34 minutes. In the San Francisco Bay area the duration was two hours and 31 minutes.

During the two weeks following the eclipse the staff of the Division of Ophthalmology of the University of California saw 23 cases of eclipse retinitis. There must have been many more cases observed by other ophthalmologists and many more persons must have had light attacks that cleared quickly and therefore were not seen.

Most of the cases occurred in children between the ages of 11 and 15 years, although there were also some adults. All gave the history of loss of vision following the observance of the eclipse without protective glasses or film. Vision at the time of examination ranged from 15/200 to 20/30. Examination of these patients revealed that many of them had a central scotoma, while in some instances a relative scotoma was present.

On fundus examination the macular area showed an edema which in two of the cases corresponded in shape to the actual eclipse. In one patient the retina had the appearance of a central angiospastic retinopathy. At the end of two weeks, vision had returned to normal in most instances with no appreciable fundus changes. In one boy, aged 13 years, vision has remained at 20/40 and the appearance of the macula in each eye suggests an early hole in the macula. In another patient, a Negress, vision has remained at 20/70 in each eye and the macula has the appearance of an early hole in the macula.

Most of the patients were unaware of the possibility of damage to the eyes from looking at the eclipse without the proper protection. In one instance, a boy, aged 11 years, looked at the eclipse while at school under the direction of the teacher. He was given some sort of dark glasses but apparently did not use them all of the time. Some of the patients had the belief that an ordinary pair of so-called dark glasses was sufficient protection. In any event, it was apparent that the laity has not been sufficiently warned of the dangers of eclipse retinitis.

"Eclipse burns" are not new and have been observed many times. During the last war they were seen a number of times among Navy personnel. It was usually observed following the use of the long glass, a monocular telescope of 20 times magnification. The burns usually resulted from "following an enemy plane into the sun." There was pain at the time of the exposure followed by loss of central vision and subsequent formation of a typical hole in the macula.

The process results from the passage of infrared rays through the layers of the retina to be absorbed by the pigment layer, where they are degraded into heat and may produce a thermal lesion. This is accompanied by swelling and edema of the retina, together with congestion of the underlying choroid, and frequently leaves a permanently pigmented scar. Probably the most extensive work on this subject is that of Verhoeff and Bell* who showed that the effects known as eclipse blindness are wholly thermic and are due to the intense concentration of the solar energy upon the retina by the refractive system of the eye itself.

Clinically the immediate effect in the milder cases is a marked scotoma which does not pass away promptly but leaves more or less "serous cloudiness" of vision that may last a few weeks. In the severer cases the scotoma is commonly central and generally of small extent, in a large percentage of cases corresponding fairly well to the dimensions of the sun's image. Wide variations from this may result due to repeated fixations overlapping. Metamorphopsia sometimes appears. In the milder cases, the scotoma tends to contract with the lapse of time, and normal vision is regained within some weeks; whereas, in the more severe cases the scotoma and loss of vision are per-

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* Verhoeff, F. H. and Bell, Louis. Proc. Amer. Arts and Sci. 51:630-811 (July) 1916.

manent. Experimentally Verhoeff and Bell were able to show that the size of the lesion is 3 mm., corresponding in size to the size of the sun's image on the retina. They also give the critical time for the development of eclipse blindness as one minute or less. Based on the cases seen among Navy personnel, it would seem that the use of the "long glass" apparently very materially shortens the time of exposure necessary to produce eclipse blindness.

Eclipse blindness is discussed in most of the textbooks but in almost none of them is there any description of the proper precautions to be taken to avoid damage to the eyes. Duane in Fuchs's textbook states that eclipse blindness occurs in patients "who watched an eclipse with glasses which were not sufficiently smoked." No mention is made of what constitutes a "sufficiently smoked" glass.

An eclipse of the sun is usually described in the press a number of days in advance so that the appearance of the phenomenon is well known. In view of the experience in San Francisco and other localities it would seem the duty of the ophthalmologists of a community to issue a warning through coöperation of the local county medical society and the press. These warnings could also describe the necessary precautions. It has also been suggested by a member of the staff that the National Society for the Prevention of Blindness or some similar organization might make it its duty to keep posted on the appearance and location of eclipses of the sun. The society could send press releases to the communities involved, describing the dangers and giving the proper precautions to be employed.

Above all it would seem that teachers should be warned of the dangers so that their pupils would not be exposed to the possibility of permanent loss of vision.

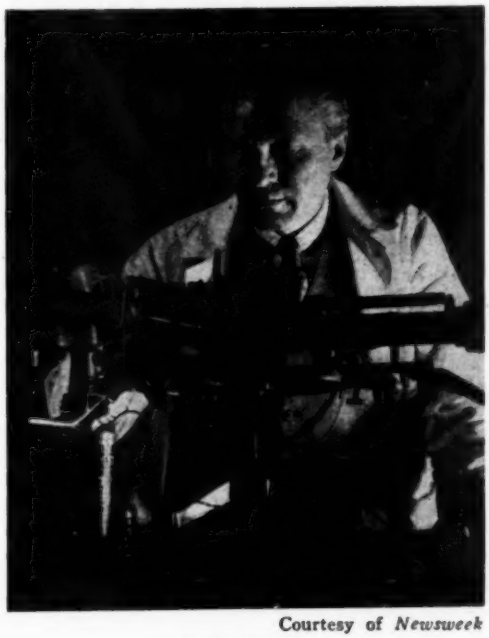
It is hoped that this editorial may stimulate some organization to give this problem its attention.

Frederick C. Cordes.

OBITUARY

SELIG HECHT
(1892-1947)

On September 18, 1947, the scientific world sustained an irretrievable loss in the untimely and sudden death of Selig Hecht, a distinguished investigator and teacher, and professor of biophysics in Columbia University. Dr. Hecht's achievements in the



Courtesy of *Newsweek*

Selig Hecht

field of general physiology and, in particular, visual physiology, rightfully gave him the distinction as a world authority on visual function.

Dr. Hecht was born in Glogow, Austria, on February 8, 1892, and was brought to this country by his parents when he was six years of age. He attended public school in New York City and later the Townsend Harris Preparatory School before entering the College of the City of New York where, as a student, he early displayed that rare intellectual curiosity and scientific ability which so characterized his entire career. Before the completion of his college course, he served as research assistant for

the United States Bureau of Fisheries. After receiving his B.S. degree he was appointed for a year as assistant pharmacologist in the Bureau of Chemistry of the United States Department of Agriculture. He then undertook graduate work at the Harvard Zoological Laboratory, receiving his Ph.D. degree in 1917. During his graduate years, he was an Austin teaching fellow at Harvard University. The summer following the receipt of his doctorate he conducted scientific research at the Scripps Oceanographic Institute at La Jolla, California. In the fall of 1917 he went to Omaha, Nebraska, where he served for four years as assistant professor of physiology in the Creighton Medical College. During the summers of these years, he carried on intensive investigations at the Marine Biological Laboratory at Woods Hole, Massachusetts. His researches there were concerned largely with the photosensitivity of the mollusca. These studies laid the basic foundations for his later investigations which dealt primarily with the photochemistry of human vision.

In 1921-22, Dr. Hecht went to England on a National Research fellowship and became an investigator in E. C. C. Baly's Spectroscopy Laboratory at the University of Liverpool. Upon return to this country, he was associated for two years with the late Prof. Lawrence J. Henderson in the laboratory of biochemistry at Harvard University. With the aid of a Rockefeller Foundation fellowship, he again went abroad, spending the year, 1924-25, at the zoological station in Naples, Italy, and the following year in Barcroft's Laboratory in Cambridge, England. In 1926, he was appointed associate professor of biophysics in Columbia University and, in 1928, was promoted to full professorship of biophysics, which position he held until his death.

The broad training in physics, chemistry, and biology that he received in this country and abroad equipped him most admirably for his scientific career. Physics and chemistry were precision tools which he used most effectively in his investigations on the kinetics of retinal photosensory processes. His bibliography, consisting of 116 publications, is concerned largely with the problems of vision in man and in animals. He wrote extensively upon many fundamental aspects of the subject, such as retinal adaptation to changing illumination, visual acuity, color vision, and the like. In recent years he became intensely interested in Planck's quantum theory of light as applied to human vision. In this connection he measured the number of light quanta necessary to produce a minimum stimulation of the human retina. As national Sigma Xi lecturer in 1944, he lectured on this subject in many American universities under the title "Energy and Vision."

During World War II, he was active in many projects concerned with vision as related to the armed forces. He was responsible investigator for various contracts for the armed forces and the O.S.R.D. He was a member of the Army-Navy O.S.R.D. Vision Committee which, after the war, became the Army-Navy National Research Council Vision Committee. Recently he lectured before the National War College in Washington on the subject "Scientific Discoveries and Social and Military Needs."

Dr. Hecht was also made a member of the Emergency Committee of Atomic Scientists, a group of nine scientists headed by Dr. Albert Einstein and formed for the purpose of acquainting the public with the problems of atomic energy. In this connection his recent and timely book *Explaining the Atom* has received high praise as a popular exposition of nuclear energy. In an editorial tribute to Professor Hecht for this book, the *New York Times* said, "We need more research scientists and teachers like him—men who are as brilliant in the laboratory as they are in the classroom or on the printed page, and, above all, men who have so little academic snobbery in them that they consider it a duty, as Clark Maxwell did in his time, to talk of science to the

common people in simple homely figures of speech that they can understand."

Dr. Hecht did possess that rare ability to discuss highly technical data in such fashion as to make the subject intelligible and stimulating to practically any audience. If he was forced to employ equations, he made sure the audience understood what the equations meant. As a lecturer he was dramatic. His subject matter was always presented with excellent logic or, may I say, with logical excellence. To listen to him develop a subject for an hour was indeed an aesthetic pleasure.

Despite Dr. Hecht's devotion to his laboratory and the many problems that constantly bothered his searching mind, he graciously shared his time for the benefit of others. He was a member of the editorial board of the *Columbia Biological Series,* the *Biological Bulletin,* the *Monographs on Experimental Biology,* and the *Journal of the Optical Society of America.* He gave freely of his time to lecture on various biologic subjects at the New School for Social Research in New York City. Recognition of his scientific achievements came to him in many forms. In 1941, he received the Frederick Ives Medal from the Optical Society of America, and, in 1942, the Townsend Harris Medal from the alumni of the College of the City of New York. He was a fellow of the American Association for the Advancement of Science, a member of the National Academy of Sciences, American Society of Zoologists, American Society of Naturalists, American Physiological Society, Optical Society of America, Harvey Society, Phi Beta Kappa, and Sigma Xi.

Although Dr. Hecht possessed a high degree of individualism, he was never a lone worker. He gave a great deal of time and energy to his graduate students, who received excellent training under his scientific leadership. A number of his students have taken prominent academic positions in the field of general physiology. Not only is his passing a tragic loss to the scientific world, but as acting President Fackenthal of Columbia University so aptly expressed it, "He will be missed by many people, in many ways."

Samuel R. Detwiler,
Professor of Anatomy,
Columbia University.

CORRESPONDENCE

The search for aqueous veins

Editor,
American Journal of Ophthalmology:

The following may answer questions raised by some visitors to my aqueous-veins exhibit at the October meeting of the Academy of Ophthalmology, concerning the difficulties connected with the search for aqueous veins.

In 1941, I found them in more than 26 percent of the patients examined with the Bausch and Lomb corneal microscope (American Journal of Ophthalmology, **25**:32, 1942). But even with a simple 10-

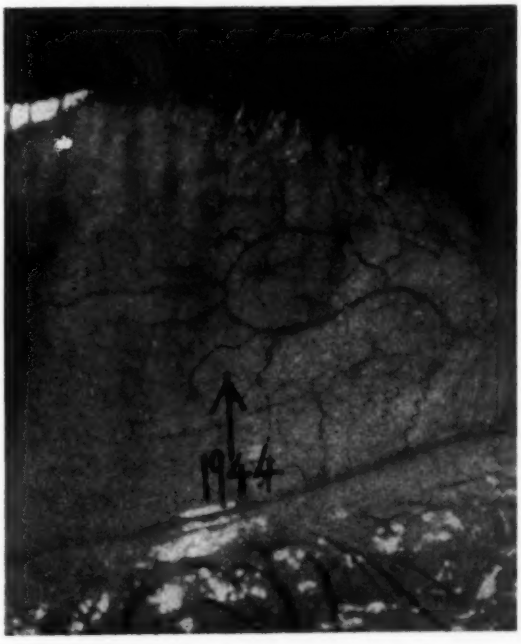


Fig. 1 (Ascher). An aqueous vein and its emptying into a stratified vessel. (5×)

times magnifying loupe, under good flashlight illumination, 14 percent of all normal eyes will show at least one aqueous vein (American Journal of Ophthalmology, **29**:1384, 1946).

With 20- to 40-times magnification, and good illumination with not too narrow a

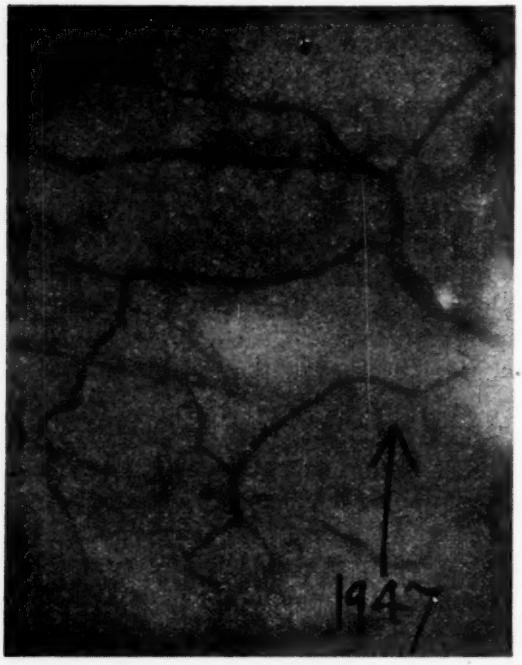


Fig. 2 (Ascher). A picture of the same vein taken 35 months later. (20×)

beam, aqueous veins are found more often. Goldmann (Ophthalmologica, **111**:147 1946) saw them in 75 percent of all normal eyes; De Vries (De zichtbare afvoer van het kamerwater, Drukkerij Kinsbergen, Amsterdam, 1947) examined 113 patients and found in all of them at least one aqueous vein; he, however, devoted to the study of one patient—including photography—as much as two hours and confessed that in some cases the search for an aqueous vein was comparable to the search for a needle in a haystack.

While I thought that the majority of aqueous veins were located near the nasal and temporal horizontal meridian, Goldmann found most of them in the temporal lower and nasal lower quadrants and De Vries counted, among 409 aqueous veins, 168 in the nasal lower quadrants.

Here, I should repeat some of the diagnostic characteristics described five years ago (American Journal of Ophthalmology, **25**:1183, 1942): The origin is in or near the limbus, often with a U-like or figure-6-like arch, or from the limbal capillaries, or—these are the largest and deepest aqueous veins—from a scleral emissary. The color of the aqueous veins is paler than that of regular veins—sometimes it is as clear as water—and often strata of different shades are seen running parallel to each other along the vessel wall. The width of aqueous veins is approximately between 0.01 and 0.1 mm. and their length varies between fractions of millimeters and one centimeter or slightly more.

Having published numerous drawings of aqueous veins, I think I should add here two photographs showing one aqueous vein and its emptying into a stratified recipient vessel, taken from the same eye of the same patient at an interval of 35 months; both pictures are unretouched and prove the constancy of aqueous veins as anatomic structures.

(Signed) K. W. Ascher,
Cincinnati, Ohio.

Inverse cyclodialysis

Editor,
American Journal of Ophthalmology:

In the July, 1947, issue of the Journal is an article by Dr. Robert N. Shaffer about inverse cyclodialysis. In the same issue Dr. H. Saul Sugar recommends the inverse method as preferable because it tends to reduce trauma.

Blaskovics gives a complete description of the inverse cyclodialysis in his book "Eingriffe am Auge" published in 1938. A horizontal incision, 10-mm. long, is made above the insertion of the superior rectus, 10 mm. from the limbus. For fixation, Blaskovics recommends a suture through the superior rectus or a strabismus hook. The capsule is

opened directly below the insertion of the muscle and the sclera is exposed.

A strictly radial scleral incision is made at the 12-o'clock position, starting below the insertion of the superior rectus extending 4 mm. toward the limbus. The dialysis is performed exactly as described by Shaffer. If one wishes another dialysis, it is performed in the opposite direction, but Blaskovics found keratic precipitates if the dialysis was too extensive. He does not mention any difficulties in reintroducing the spatula. Maybe he was such an excellent surgeon that there was no bevel with his incision.

Since reading Dr. Shaffer's paper, I have used the inverse cyclodialysis in several cases with equally good results. The main improvements in this operation since it was originated by Blaskovics seem to me to be the following:

1. Gonioscopy for the selection of the best operative site.
2. Fixation of the eyeball with a scleral suture if the incision is not done below the superior rectus muscle.
3. Air injection into the anterior chamber to prevent or stop bleeding.
4. Postoperative position of the patient's head.

Dr. Shaffer and Dr. Sugar deserve great credit for their papers which reintroduce and modify the inverse cyclodialysis for American ophthalmologists.

(Signed) Ernest E. Hessing,
Oakland, California.

BOOK REVIEWS

GIFFORD'S TEXTBOOK OF OPHTHALMOLOGY. By Francis H. Adler, M.D., professor of ophthalmology, Universty of Pennsylvania Medical School. 4th edition. Philadelphia and London, W. B. Saunders Company, 1947. 512 pages. Price, $5.50.

This book, termed by the publisher the fourth edition of "Gifford's Textbook of Ophthalmology," has undergone such radical changes in appearance, arrangement, illustrations, and text that it might well have been offered as a completely new volume. The reputation of Dr. Adler is such that one would not have thought it necessary for the publisher to capitalize on the Gifford name while at the same time eliminating most of the personality of the original author. Carful comparison of the present edition with the third edition reveals occasional sentences and paragraphs retained in unchanged form but for the most part the text has been completely rewritten with some material omitted and a considerable amount added. Numerous new illustrations, many of them excellent, have been included.

Dr. Adler's interest in physiology has led to the inclusion of much subject matter not ordinarily found in a textbook of this scope. Considerable detail is presented on the visual pathways and 50 pages are devoted to disturbances in ocular motility. That these are accurate and well presented will be anticipated by any reader familiar with the author's (revisor's) teachings and writings but it may be wondered whether much of this material is not at the level of postgraduate teaching. On the other hand, methods of refraction and surgical procedures have been greatly minimized, as is proper in a textbook prepared ostensibly for undergraduate medical students and general practitioners. The chapters on "Ocular Manifestations of General Disease" and on "Ocular Disorders Due to Diseases of the Central Nervous System" are particularly valuable and appropriate.

Another innovation is the addition of bibliographic references at the end of each chapter. These have been somewhat arbitrarily selected and are far from complete but may suggest additional reading for the more advanced student.

Many of Dr. Gifford's special procedures in therapy, such as delimiting keratotomy and the use of trichloracetic acid in corneal ulcers, are briefly mentioned but not always as originally advocated. For example, in the use of trichloracetic acid the essential thing

is to have no excess of acid on the very fine-tipped applicator (Gifford always removed the excess by wiping on his thumb nail) and not the subsequent irrigation with normal saline as advocated in the present text. The metric system for recording visual acuity has been substituted for the "foot" system, although the latter continues to be the most widely used in this country in spite of all theoretical objections.

There is naturally a legitimate difference of opinion as to what a textbook on ophthalmology intended for use by medical students should contain. The author of any such text is obliged to reach a compromise between completeness and the brevity and concise presentation essential to his purpose. Unfortunately, the medical curriculum is so crowded that in most schools ophthalmology, the "queen of the specialties," is often relegated to an undeservedly inferior position on the part of the administrative authorities and students. The number of hours assigned for didactic and clinical work in this field are wholly inadequate for a highly comprehensive study. Students have neither the time nor the inclination, unless they possess a very unusual interest, to read voluminous treatises on the eye. The particular task of the teacher, then, is to emphasize the recognition of the more important eye diseases and particularly the relation of the eye to general disease. In correlation with this, reference books aimed at this particular educational level are essential.

As a *reference book* for undergraduate medical students and general practitioners this new edition of Gifford's well-known textbook, rewritten by Adler, is excellent and deserves wide use. The format and typography are pleasing and the material is well prepared and authentic. Its greatest value, however, lies for students at the postgraduate level who, beginning a real study of ophthalmology, seek a simplified dissertation on the eye before delving into the more complicated and extensive discussions found in the standard reference books and in the current literature. It would also not be surprising if practicing ophthalmologists found it extremely convenient and useful.

William A. Mann.

A HANDBOOK OF OCULAR THERAPEUTICS. By the late Sanford R. Gifford, M.D. Revised by Derrick Vail, M.D., D.O. (Oxon.). 4th edition. Philadelphia, Lea & Febiger, 1947. 336 pages. 66 illustrations. Price, $5.00.

In the revision of Gifford's masterpiece, Vail has blended with admirable skill the personal approach of the original author with the later advances in ocular therapeutics. Gifford's teaching has been most influential in determining modern therapeutic practice, especially among the younger ophthalmologists, and his handbook has become a recognized classic in its field. As administrator of this heritage, Vail was a natural choice both because of his long intimacy with Gifford and their common background of professional ideals and ophthalmic tradition.

Future editions, while preserving the best of Gifford's contribution, will undoubtedly reveal more of Vail's own personality. Revision involves the triple responsibility of creation, preservation, and destruction. In the present volume, Vail has been probably unduly conservative in adding new material or deleting old. Among the additions are recent studies in chemical burns of the cornea, the newer therapy of seborrheic blepharitis, a discussion of D.F.P. (diisopropyl fluorophosphate), typhoid vaccine for vasodilatation, and the differential treatment of thyrotoxic and thyrotropic exophthamos. Agents whose place in ocular therapeutics is not yet established, such as rutin, the antihistamin drugs, and the newer mydriatics have been judiciously omitted.

Some new ideas that might have been included are glycerine for clearing the cornea in acute glaucoma to permit ophthalmoscopy, the diagnostic patch test for tuberculosis,

BAL (2,3- dimercaptopropanol) for the elimination of arsenic in tryparsamide and other reactions, gelfoam and thrombin for hemostasis, and merthiolate and zephiran as alternative preservatives for ophthalmic solutions. In the discussion of sulfonamides, their mechanism of action is insufficiently detailed, sulfapyridine might well be omitted, and sulfadiazine given much more emphasis. Sulfacetimide is no more than listed. The text reflects the previous need for economy in the use of penicillin, but the present tendency is toward massive concentration in subconjunctival injections and in ophthalmic ointments. The deteriorating action of adrenalin on penicillin is more than counterbalanced by the prolonged localization that its proper use insures.

The book deals brilliantly with office surgery, but neglects attention to some aged but excellent procedures, such as cautery puncture for ectropion, entropion, and lymphangiectases, Agnew's incision for acute dacryocystitis through the cul-de-sac, and Crigler's compression maneuver for congenital impatency of the nasolacrimal duct.

Each edition of Gifford's *Therapeutics* has been a significant improvement over its predecessor, and the present edition meets the challenge worthily. This slim volume, though crammed with information, is written with an effortless style that makes cover to cover reading as enjoyable as the contents are profitable.

James E. Lebensohn.

FUNDAMENTALS OF CLINICAL NEUROLOGY. By H. Houston Merritt, Fred A. Mettler, and Tracy Jackson Putnam. Philadelphia, The Blakiston Company, 1947. 289 pages, 96 figures, index. Price, $6.00.

Ophthalmologists, as a general rule, confess to some weakness in neurology. Most of us are aware of the neurologic conditions that lie within our field, but not many are truly expert in this branch of medicine that is so important to ophthalmology. The authors have given us a much needed tool with which to work. The volume is beautifully illustrated and executed. It covers the entire field of clinical neurology in a most economical yet lucid fashion, and the reputations of the authors vouch for its accuracy. Ophthalmologists will find it most useful in refreshing their recollections of neurologic disorders, and valuable in the interpretation of problems met with in their daily practice.

Derrick Vail.

DISEASES OF CHILDREN'S EYES. By James Hamilton Doggart, M.D., St. Louis, C. V. Mosby Co., 1947. Clothbound, 288 pages, 210 illustrations, including 32 colored plates. Price, $10.00.

In his preface, the author disclaims any special knowledge about this particular phase of ophthalmic practice. He also states that he did not have a large clinic on which to draw for material, but because of his wartime assignment he had become interested in the subject and had thought that collecting in one book the material from previous publications that might be considered particularly applicable might serve a useful purpose. The reviewer thinks that he has done this satisfactorily but that all of the material is available in the best textbooks. It is hard to understand the need for a book devoted exclusively to the eyes of children. However, if one desires a book confined to the problems of this subject, he might add this volume to his library. It is well prepared, of an acceptable size, in good print with good illustrations and a substantial binding, characteristic of the products of this publisher.

Lawrence T. Post.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Haden, Henry C. **The development of the ectodermal framework of the optic nerve, with especial reference to the glial lamina cribrosa.** Am. J. Ophth. **30**:1205-1214, Oct., 1947. (15 figures.)

Wilczek, Marian. **The lamina cribrosa and its nature.** Brit. J. Ophth. **30**:551-565, Sept., 1947.

Since the work of Fuchs in 1916 there has been little research on the nature of the lamina cribrosa. Fuchs stated that some of the internal fibers of the sclera extend through the nerve fibers across the foramen sclerae and form the sieve that gives this membrane its name. Wilczek made detailed studies by means of serial sections and wax plate reconstructions of the area of the membrane in fetal, post-natal, and adult life. He found that the lamina is composed of neuroglia fibers from the dural and pial coverings of the optic nerve as well as from scleral fibers. It is not an independent structure but is simply the strongest part of the septal system of the nerve. The function of the lamina is essentially the fixation of the nerve in the scleral canal where it is subject to danger of traction. The serial sections proved, in addition, that at the level of the lamina the nerve bundles are subject to the most constriction and hence are most easily damaged by inflammatory processes, edema and intraocular pressure. (14 figures.) Morris Kaplan.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Braley, A. E., and Alexander, R. C. **Virus studies in lymphomatoid diseases of the ocular adnexa.** Am. J. Ophth. **30**: 1369-1380, Nov., 1947. (4 figures.)

Brown, A. L., and Nantz, F. A. **Corneal wound healing II: variations in adhesive power of fibrin. In vitro studies** Tr. Am. Ophth. Soc. **44**:85-92, 1946.

The authors present experimental evidence of the adhesive power of fibrin in the cycle of corneal healing in the rabbit and dog. By an ingenious method employing standardized corneal strips and various concentrations of fibrin the ten-

sion of the adhesive power of fibrin in experimental wound healing was accurately measured under varying conditions. C. D. F. Jensen.

Carsten, M. E. **A rapid procedure for the embedding of eyes.** Arch. Path. **44**: 96-100, July, 1947.

Preparing eyes for microscopic study is a very slow process. Rapid methods of dehydration and embedding cause shrinkage and consequent distortion. Where rapid diagnosis is called for and experimental work is being done a water soluble embedding medium which would not shrink or dehydrate tissues was being sought.

The author describes a method of embedding eyes by means of water-soluble "carbowax" (polyethylene glycol) and "carbowax" ester used in succession. There is no shrinkage, dehydration, or fungus growth associated with the technique. The tissue is well preserved.

By this method sections can be cut as thin as 7 microns, but for easy mounting 10 to 15 microns is preferable.

Francis M. Crage.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Aldrige, W. H., Davson, H., Dunphy, E. B., and Uhde, G. I. **The effects of di-isopropyl fluorophosphate vapor on the eye.** Am. J. Ophth. **30**:1405-1412, Nov., 1947. (1 figure.)

Ferraro, A., Roizin, L., and Givner, I. **Ocular changes in rats on diets deficient in amino acids. II. Corneal dystrophy due to valine deficiency.** Arch. Ophth. **38**: 342-352, Sept., 1947.

In this preliminary communication, the authors report observations on rats reared on a valine-deficient diet. Changes develop in the cornea, in addition to general changes and structural alterations in other organs, that seem to be the result of edema and progressive degeneration, and gradually lead to keratinization and disorganization of the epithelium of the cornea. These changes in the eye appear to be reversible if treated in time. (6 figures, 11 references.)

R. W. Danielson.

Ferraro, A., and Roizin, L. **Ocular involvement in rats on diets deficient in amino acids. I. Tryptophan.** Arch. Ophth. **38**:331-341, Sept., 1947.

During the past four years, the authors have been studying the effects of various diets deficient in amino acid on the growth, development and morphology of various tissues and organs, endocrine glands and nervous system in rats. Ocular involvement occurred with diets deficient in tryptophan and valine. Tryptophan is able to prevent, improve and cure the general clinical, as well as some of the ocular symptoms produced experimentally by tryptophan-deficient diets. These changes are of two types. The earlier changes characterized chiefly by water splitting of the lens fibers and vacuolation were found to be reversible under tryptophan treatment. The more advanced changes which lead to disorganization of the structure of the lens seem to be irreversible. (4 figures, 7 references.)

R. W. Danielson.

Gillette, D. F. **Visual disturbances due to digitalis.** Tr. Am. Ophth. Soc. **44**: 156-165, 1946.

The author reviews the literature concerning the types of visual disturbance that can result from mild and more severe forms of digitalis intoxication. He presents three interesting cases of digitalis poisoning, two of which were characterized by central scotomata and diminished visual acuity. Withdrawal of the drug

permitted the relatively prompt return of normal vision. In each case several types and degrees of chromatopsia are described. C. D. F. Jensen.

Matteucci, P. **Sympathetic innervation and neuro-vegetative regulation of the uvea.** Rassegna ital. d'ottal. **16**:283-292, July-Aug., 1947.

Animal experimentation leads the author to several conclusions regarding the nerve control of the uvea. Rapid degeneration of the vegetative fibers of the iris after resection of the ciliary nerves indicates that the iris contains no nerve ganglia. The caroted sinus, with its sympathetic, vagal and sensory innervation is an important regulatory center of uveal circulation. Uveal vasomotor fibers do not all stop in the superior cervical ganglion; some follow the pericaroted sympathetic. The influence of the cervical sympathetic upon ocular tension may be a result of its control of capillary tone. Eugene M. Blake.

Robbie, W. A., Leinfelder, P. J., and Duane, T. D. **Cyanide inhibition of corneal respiration.** Am. J. Ophth. **30**:1381-1386, Nov., 1947. (4 figures.)

von Sallmann, L., and Dillon, B. **The effect of di-isopropyl fluorophosphate on the capillaries of the anterior segment of the eye in rabbits.** Am. J. Ophth. **30**: 1244-1262, Oct., 1947. (8 figures, 43 references.)

Scuderi, R., and Morone, G. **Action of vestibular stimulation on the pressure in the central retinal artery.** Riv. di oftal. **2**:22-36, Jan.-Feb., 1947.

In 25 persons between 14 and 30 years of age, the retinal arterial diastolic pressure was measured before and 15, 30, 45, 50, 90 seconds, 2, 3, 4, 5, and 10 minutes after the caloric and rotary stimulation of the labyrinth. Both the caloric test of Veits and the rotatory test of Barany regularly produced a decrease of the intraärterial pressure, amounting to five grams (Baillart) after the rotatory tests and seven grams after the caloric test, and lasting for two to three minutes. The drop in arterial pressure always occurred in both eyes and was of the same duration as the nystagmus which accompanied the tests. The decrease in arterial pressure in the retinal arteries is not due to a drop in general blood pressure. K. W. Ascher.

Sloan, L. L., and Gilger, A. P. **Visual effects of tridone.** Am. J. Ophth. **30**:1387-1405, Nov., 1947. (25 figures.)

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Borsello, G., and Gomirato, G. The adaptation curve and the visual field behavior to normal and attenuated light in neurasthenic constitutional syndrome. Riv. Oto-neuro-oftal. **21**:278-311, Sept.-Dec., 1946.

In 14 neurasthenic patients between 25 and 35 years of age dark adaptation was retarded and reached terminal values inferior to normal. The visual fields in normal light showed a slight concentric contraction for green. In dim light the chromatic field was very much more contracted than in normal subjects. The authors discuss the probable pathogenesis of these changes concluding that they are in part a local result of the general neuro-vegetative-endocrine changes and in part an expression of a miopragic constitution of the retina. (15 figures, bibliography.) Melchiore Lombardo.

Boström, C. G., and Kugelberg, I. **Official color sense control in Sweden.** Arch. Ophth. **38**:378-380, Sept., 1947.

This paper describes the development and use of the authors' color sense tests in Sweden. R. W. Danielson.

Clark, W. E. Le Gros. **A possible central mechanism for color vision.** Nature 160:123-124, July 26, 1947.

The author suggests that the correlations in this article (together with those reported in a previous paper) offer further presumptive evidence that the six-layered pattern of the geniculate nucleus (that is, two sets of three layers for the two eyes) may be related to color vision. However, further and more detailed correlations need to be established, and much more experimental evidence is required in order to put this thesis to a crucial test.

Theodore M. Shapira.

Dubois-Poulsen, A., and Rozan, A. **Graphic study of accommodative fatigue.** Ann. D'Ocul. 180:206-237, April, 1947.

Made partly from a toy railroad and track, the apparatus used is a modification of that used by Howe and Berens-Stark. It consists essentially of a continuous cord tracking on several pulleys which is attached to a square vertical frame. To the cord a target and a recording stylus are attached. The examinee by pulling the posterior vertical part of the cord down simultaneously moves the target nearer his eye and the stylus proportionately up.

For 195 office patients records were made for three minute periods with oscillation periods of approximately 45 to 60 seconds, in an illumination of 8 to 14 lux. In 30 percent accommodative fatigue was observed during the test. In 40 to 50 percent the accommodation remained constant, and in 28 percent the accommodation increased during the test. In monocular fixation fairly marked variations were observed between the static and the dynamic near points. This difference was increased by fatigue and was roughly proportionate to the rapidity of contraction of the ciliary muscle. Relatively little variation occurred in the same subject on repeated tests. With binocular vision the curves were essentially similar except that the decrease of the dynamic near point was less accelerated. Accommodative fatigability changed little with age or sex or with quantitative and qualitative variations of illumination within reasonable limits. In hypermetropic and astigmatic subjects accommodative fatigue was greater than in the mypopic. General bodily exhaustion was observed to be a more important factor than muscular fatigue. The results obtained following the instillation of parasympatheticomimetic and sympatheticomimetic drugs, and in hyperemia and increased ocular tension were not conclusive although it is probable that increased accommodative fatigability is frequently an early sign of ocular hypertension. (13 figures, 13 references.)

Chas. A. Bahn.

Gernandt, B., and Granit, R. **Single fiber analysis of inhibition and the polarity of the retinal elements.** J. Neurophysiol. 10:295-302, July, 1947.

This article is not readily abstracted to a form shorter than its summary which appears below. A condensation of this article may be found in Nature 159:806, 1947.

"Single fiber responses have been isolated from the nasal region of the cat's retina with the micro-electrode technique and tested by illumination and polarisation of the retina between fixed electrodes.

The pure on-elements respond to threshold cathodal polarisation with an on-effect. An increase of current strength by some 30 percent elicits a threshold anodal off-effect.

The pure off-elements (inhibited by

light) have opposite polarity. They respond to cathodal polarisation at the threshold with an off-effect, to anodal stimulation of the same strength with an on-effect (not elicitable by light).

The on-off-elements are either "cathodal" or "anodal", depending upon their off/on-ratio determined by illumination at threshold strength. They are held to be combinations of on-paths and off-paths.

The results form the basis of a simple explanation of the nature of inhibition in the retina in terms of the horizontal and amacrine cells." Elek Ludvigh.

Gernandt, B. **Colour sensitivity, contrast and polarity of the retinal elements.** J. Neurophysiol. **10**:303-308, July, 1947.

This article is not readily abstracted to a form shorter than its summary which appears below. A condensation of this article may be found in Nature **159**:806, 1947.

"The micro-electrode technique and the cat's retina have been used for a first analysis of the spectral sensitivity of well-isolated retinal elements during anodal and cathodal polarisation. It was found that, depending upon the polarity, different spectral regions were enhanced or depressed so that polarisation proved a convenient and very direct method of color analysis.

The on-elements were sensitive to short wave-lengths only, the off-elements to both long and short wave-lengths; the on-off-elements had humps or depressions in various regions. The regions in which enhancements or depressions chiefly occurred were (in the order enumerated) 0.520, 0.600, 0.470, and 0.570μ, the humps in 0.600μ being particularly large.

The on- and off-components of the isolated spike of an on-off-element were often differentially sensitive to "contrasting" regions of the spectrum, suggesting that the two components are running in different but adjacent and well-synchronized fibers."

Elek Ludvigh.

Hartridge, H. **Recent advances in the physiology of vision.** Brit. M. J. pp. 913-916, June 28, 1947.

The author uses retinal micro-stimulation to form geometric images of a diameter equal to one-fifth of a cone unit. A green ray (5,200 A.U.) moved less than one-fourth of a cone unit on repeated tests. Different fixation points were found for the following colors: red, orange, yellow, green, blue green, greenish blue and blue. A white beam remained white wherever it was situated on the retina unless conditioning colored lights were present in the visual field. The white then took on all seven previously mentioned colors. Color mixture experiments showed that the seven colors could be produced when various proportions of the others were mixed. Young's theory of color vision is discussed in the light of the seven color receptors.

From the response curves and tests for hue discrimination, the author concludes there are at least five types of receptors for color in the retina. There is a loss of color vision normally when reducing the visual angle or the illumination is reduced in peripheral vision. Colored objects which subtend small angles often become altered and appear to be of another color. The author concludes that there are at least seven receptors for color in the human retina. (11 figures.)

H. C. Weinberg.

Kinsey, V. E. **Transfer of ascorbic acid and related compounds across the blood-aqueous barrier.** Am. J. Ophth. **30**:1262-1266, Oct., 1947. (5 figures, 4 references.)

Mann, Ida. **Contact lenses.** Brit. J. Ophth. **31**:565-568, Sept., 1947.

To the newly formed Contact Lens Society, Mann delivered this presidential address as a plea for the accumulation of more data and the planning of further experiments. The problem of contact lenses is unusual in that animal experimentation is almost impossible because visual acuity and subjective tolerance are not measurable. The problems of prisms, of cylinders and of bifocals still demand much work.

The most pressing problem is that of tolerance. Mann sent a questionnaire to 100 patients for whom she had fitted lenses without subsequent control of data. Of 61 who had myopia 11 had an error of 5.00 diopters or less, and the others up to 22 diopters. Ten patients who were given one lens only for monocular aphakia did not wear it. Of 11 who had conical cornea 9 wore the lens all day, and none was a complete failure. The patients were all asked if, knowing what they know now, they would go in for contact lenses again. Sixty-four replied unhesitatingly yes and 19 gave an equally emphatic no. Seventeen said if they could be made more comfortable they would say yes. Must the lenses be worn every day for good tolerance? Fifty-five wore their lenses every day, 47 had enough tolerance for a full day's work and 31 wore them more than 12 hours every day. On the other hand 9 wore them only occasionally with excellent tolerance so that daily wear was not considered necessary. Tolerance could not be associated with any special condition of the eyes, except that in conical cornea contact glasses were almost always successful and in monocular aphakia always useless.

To the questions concerning solutions, 49 answered that they insert their lenses dry, 22 patients used normal saline and 9 used tap water. Obviously much has to be learned about solutions and much has to be done for the whole problem of contact lenses. Morris Kaplan.

Mann, I., and Sharpley, F. **The normal visual (rod) field of the dark-adapted eye.** J. Physiol. **106**:301-304, July, 1947.

The rod visual field of the completely dark-adapted eye was mapped out within 90° limits from the fixation point for 33 normal individuals. The mean angular radius of the plotted fields for 80 subjects (including 47 previously tested) is given by age groups, and a tendency for the field to contract with advancing age is observed. The possible reasons for this contraction with age are discussed.

Theodore M. Shapira.

Matteucci, P., and Carucci, E. **Mydriasis and acute intraocular hypertension.** Ann. d'ocul. **180**:360-365, June, 1947.

In rabbits, mydriasis is not primarily produced by acute ocular tension. Pupillary dilatation is basically caused by a reflex parasympathetic photomotor paresis or by hyperfunction of the sympathetic control. In a first group of rabbits, ligature of vorticose veins was performed at once; in a second group retrobulbar injection of nicotine preceded the ligation of the vorticose veins. In a third group the ciliary nerves were resected, then the vorticose veins were ligated. After the vorticose vein ligation the tension rose from 20 to 50 mm. Hg during the first day and by the fifth day decreased to approximately 15. The pupil size remained at 4 mm. practically constantly. There was also no parallelism between the ocular tension and the pupillary size in the other two groups. (4 figures, 15 references.) Chas. A. Bahn.

Pascal, J. I. **A radical innovation in contact lenses.** Arch. Ophth. **38**:381-382, Sept., 1947.

After discussing the history of attempts to make contact lenses comfortable, Pascal says that an experimental study as to what kind of curve would best fit the great majority of eyes brought to light the almost unexpected emergence of the cone. It was found that if the scleral section was made conical, and the angle of the cone was so chosen that it rested tangentially against the eyeball a minimum of pressure was exerted. Instead of a large bearing surface between lens and sclera, it was found that sometimes a narrow tangential band one to three mm. wide set inside the edge of the lens gave more comfort than any other surface.

R. W. Danielson.

Pascal, J. I. **The power or powers of a lens.** Brit. J. Ophth. **31**:570-572, Sept., 1947.

The method of calculating reduced surface power of the crystalline lens is simplified by what is called the "dam" formula: $D = aM$. In this formula a is the difference between the two indices and M is the curvature in metrecs which is obtained by dividing the radius in mm. into 1000.

Morris Kaplan.

Rezende, Cyro de. **Mirror writing.** Arq. brasil. de oftal. **10**:20-31, 1947.

The author quotes a number of previous writers on the subject, and then records and illustrates a personal case, in a boy of eight years. Other conditions were normal. The vision of the right eye was 0.2 that of the left eye 1.0, and the refraction was apparently normal. The mirror writing involved words and drawings, but not numbers. (References; an excellent full-page illustration of the peculiarity discussed.)

W. H. Crisp.

Rose, H. W., and Schmidt, I. **Factors affecting dark adaptation.** J. Aviation Med. **18**:218-230, June, 1947.

A survey of the literature on factors affecting dark adaptation is reported. Experimental data are presented that show that caffeine-metrazol (caffeine .05 gm., metrazol .1 gm.), mild muscular exercise, strychnine nitrate .005 gm., ultrasonic vibrations of 24,000 c.p.s., .003 gm. fervitin, .05 gm. ephedrine hydrochloride, .05 gm. octin D, stimulation of taste with saccharin, or the administration of 266,000 I. U. of vitamin A in emulsion or oil daily for sixteen days, do not significantly affect the dark adaptation of normal subjects.

Elek Ludvigh.

Sebas, S. R. **Optical correction of astigmatism.** Rev. brasil de oftal. **6**:17-24, Sept., 1947.

The author's routine method includes the clock dial, the ophthalmometer, skiascopy, and the use of test lenses as described by Donders ninety years ago. Cases of "spasm of accommodation," he would refer to the neurologist before undertaking the measurement of refraction. He has apparently little respect for the use of cyclopegics in the measurement of astigmatism.

W. H. Crisp.

Temple Smith, E. **Thoughts on refraction.** M. J. Australia **2**:69-71, July 19, 1947.

Cycloplegics should never be used as a routine measure, but only when indicated. In children atropine is indicated because of the amazing faculty the child has for suppressing hypermetropia and in young adults cycloplegics should be used when ciliary spasm is suspected. A subjective examination is necessary in presbyopic adults to check the retinoscopy. An examination in cycloplegia necessitates two or three visits with loss of time, and causes annoyance to the business man. Mydriasis is obtained by using a mixture of cocaine, adrenalin, and neo-synephrine.

Complete dilatation of the pupil, in the presence of small errors, causes confusion by giving conflicting retinoscopic reflexes, but should be used to examine the extreme periphery of the retina.

H. C. Weinberg.

Veasey, C. A., Jr. **The dissatisfied refraction patient.** Am. J. Ophth. **30**:1286-1293, October, 1947.

5

DIAGNOSIS AND THERAPY

Adrogué, E., and Wolf, J. A. **Gonioscopic examination; its technique with Goldman's contact glass.** Arch. chilenos de oftal. **3**:305-312, Nov.-Dec., 1946.

The authors remark that so recent a work as that of Berens and Zuckermann on diagnostic examination of the eye does not mention Goldman's procedure. The various details of the technique for examination according to Goldman are carefully explained, under the headings of previous adjustment of the lamp, placing of the contact glass, and the actual examination, as well as interpretation of the image in optical sections. In conclusion the authors discuss the principal difficulties which may occur during the gonioscopic examination; including irritability of the eye calling for repeated anesthetization, occasional inversion of the eyelids causing appearance of the lashes in the gonioscopic image, absence of the necessary layer of liquid between cornea and the gonioscopic lens, excessive obliquity of the source of illumination, and the development of corneal edema. (2 figures, references.) W. H. Crisp.

Bellows, J. G., and Farmer, C. J. **Streptomycin in ophthalmology.** Am. J. Ophth. **30**:1215-1220, Oct., 1947. (3 figures, 9 references.)

Calogero, V. N. **Rubrophen treatment of ocular tuberculosis.** Riv. di oftal. **2**:58-69, Jan.-Feb., 1947.

Rubrophen, a triphenylmethane derivative, was introduced into therapy by Sailer, in 1937. For extrapulmonary tuberculosis, it is used either in comprettes containing 0.15 gram, or intravenously in doses of 0.30 gram with the addition of 0.08 gram of sodium bisulfite. Five percent ointment can be administered in a lanolin base. The therapetutic dose of 0.30 is 1/150 of the fatal dose of 50 grams for a patient who weighs 70 kilograms. In five patients suffering from anterior segment tuberculosis and observed for many years the application of the rubrophen, oral and parenteral, proved beneficial. (Bibliography.) K. W. Ascher.

Chamlin, Max. **Technical methods for the 1/2,000** field. Am. J. Ophth. **30**:1414-1423, Nov., 1947. (3 figures.)

ten Doesschate, J. **Perimetric charts in equivalent projection allowing a planimetric determination of the extension of the visual field.** Ophthalmologica **113**:257-270, May, 1947.

As stated in the title, the purpose of this study was to design perimetric charts that would lend themselves to planimetric measurement. To meet this requirement the perimetric findings which are spatial values obtained on the inside of a sphere have to be transferred to or projected onto a plane surface in such a way that all areas enclosed by a closed line on the sphere are directly proportional to the corresponding areas on the surface of projection (the principle of equivalent projection). Since none of the existing perimetric charts meets this requirement, the author by applying spherical trigonometry, devised one in which the relationship between perimetric angle (or

angle of eccentricity) and radial distance on the chart is as follows:

perimetric angle in degrees	linear (radial) distance in units of lengths
10	4.9
20	9.8
30	14.6
40	19.4
50	23.9
60	28.3
70	32.4
80	36.4
90	40.0
100	43.3
110	46.3

Such a chart is suitable for planimetry but not free of distortion. The result of the planimetric measurement, that is a figure expressing area, is useful for statistical purposes, but does not express the practical usefulness of the visual field. A round concentric field of a certain area is doubtlessly more useful than a hemianopic field of the same area.

The author has also designed a chart in which the area of the field is a measure of the number of functioning receptor units. Such a chart, however, is clinically impractical. Peter C. Kronfeld.

Guy, L. **Dangerous explosion of transilluminator.** Arch. Ophth. **38**:382-383, Sept., 1947.

The author reports that his Lancaster transilluminator exploded when turned on. The tip hit the wall with sufficient force to break the plaster. Fortunately, the instrument had not yet been inserted behind the eyeball. R. W. Danielson.

Halbron, P., and Aitoff, H. **Local use of penicillin in external gonococcic affections of the eye.** Rev. brasil de oftal. **6**:25-29, Sept., 1947.

The authors describe seven cases in which penicillin was used locally, either in collyrium or in ointment. The collyrium contains a concentration of 5,000 Oxford units per cubic centimeter. The ointment contains 1,000 Oxford units per gram. Both preparations, kept in the refrigerator, remained effective for a week. Local treatment with penicillin of gonococcic ophthalmia represents a real advance over previous forms of therapy. W. H. Crisp.

Ingalls, R. G. **Current status of penicillin in ocular infections.** Am. Practitioner **1**:680-683, Aug., 1947.

Only those diseases caused by bacteria sensitive to penicillin are cured by the drug. As larger doses are being used, more bacteria that are sensitive are being found. Failure with bacteria usually sensitive may be due to resistance built up from inadequate doses, or to the need for high initial dosage. In the treatment of intraocular infections one should not await the laboratory findings, but should begin immediately with penicillin and sulfonamide therapy. Infected traumatic wounds of the anterior segment may be treated with penicillin solution. In vitreous infection, intravitreal injections of purified penicillin should be used. (References.) Bennett W. Muir.

Johnson, M. H. **Method for restoration of human orbits following necroscopy enucleation.** Am. J. Ophth. **30**:1298, Oct., 1947.

Magitot, A. **Tissue therapy.** Ann. d'ocul. **180**:376-378, June, 1947.

Is tissue therapy worthwhile? Based upon a résumé of the literature, the pros and cons are summarized as described by Filatov and Verbitska. The treatment consists essentially of intramuscular in-

jections of cod liver oil prepared in a specific manner and of subconjunctival and subcutaneous tissue implantations. For the subconjunctival implantations placenta is used but the subcutaneous injections consist of a varied assortment of biologic entities ranging from aloes to ovaries. In the treatment of pigmentary retinal degeneration and many other ocular affections ranging from trachmatous pannus to sclero-choroidal myopia, its usefulness is somewhat problematic. The essential factor in the treatment is apparently a new type of "Biologic Activator" which seems to be a sonorous but somewhat meaningless term. Improvements are recorded in such small units that only God and the authors can measure the progress. Chas. A. Bahn.

Sorsby, Arnold. **Penicillin in ophthalmia neonatorum.** Brit. M. J. pp. 913-916, June 28, 1947.

Penicillin was used in the treatment of ophthalmia neonatorum by a variety of methods. In thirty cases local instillation of drops containing 2500 units per ml. at five-minute intervals gave good results. In fifty-two cases these drops were given at one-minute intervals and the eyes became clear within an average time of 33 hours. Twenty-seven patients recovered within 48 hours when drops containing 10,000 units per ml. were used. In fifty-one cases the use of penicillin in an oily or solid vehicle at less frequent intervals was unsatisfactory.

Four systemic injections of 200,000 units at intervals of three hours combined with local medication was followed by recovery within 46 hours in 84 percent of patients. In infection with diphtheroid organisms or inclusion bodies treatment was not limited to penicillin. In no case has penicillin treatment failed to remove all anxiety within a few hours. (11 tables.)

H. C. Weinberg.

Sorsby, A., and Ungar, J. **Distribution of penicillin in the eye after subconjunctival injection.** Brit. J. Ophth. **31**:517-528, Sept., 1947.

Comparisons of intraocular concentrations of penicillin after various routes of application were made. Subconjunctival injections with and without adrenalin were compared with instillations of highly concentrated ointment and with intravenous injections. By far the most satisfactory therapeutic concentration was obtained by the injection of the drug in 50,000 unit doses dissolved in 0.5 cc. of 1 to 1,000 adrenalin solution beneath the conjunctiva in cocaine anesthesia every six hours. The concentration reached in all the eye tissues was several times greater in the 50,000-unit dose than with a 20,000-unit dose and was maintained considerably longer. When injections are made with adrenalin fairly substantial levels of penicillin are still present in the fellow eye at the end of 4 to 6 hours. The ointment used contained 50,000 units per gram and the intravenous injections were of 25,000 units. In both these routes, the concentrations were considerably less in all tissues than with the subconjunctival injection with adrenalin. (14 tables.)

Morris Kaplan.

6

OCULAR MOTILITY

Adler, F. H., and Jackson, F. E. **Correlations between sensory and motor disturbances in convergent squint.** Arch. Ophth. **38**:289-300, Sept., 1947.

The important factor in the production of convergent comitant squint is the primary force which turns the eyes in, i.e. the excessive convergence innervation. In only one group is one thoroughly cognizant of the manner in which an abnormal convergence stimulus is produced and how it causes squint. This is the group in

which the convergence is due to excessive hypermetropia. It is the purpose of this paper to consider the senory relationships found in all types of convergent squint and to determine the correlations between the sensory abnormalities and the motor disabilities in the hope that some light may be thrown on the source of this excessive convergence. One hundred and seventy-five private patients form the basis of this study. In this statistical analysis, the most striking feature is the relationship between the type of correspondence and the angle of squint. Persons with squints of low angle have predominantly normal correspondence, whereas persons with squints of 15 to 20 degrees and over usually have anomalous correspondence. The squints of low angle fall largely into the accommodative group; the squints of large angle belong predominantly in the class for which no causal factor is known. Age of incidence, duration of squint and presence or absence of amblyopia have little to do with determining the type of correspondence. It is possible that the cause of the squint of undetermined origin is anomalous correspondence itself. (4 references.)

R. W. Danielson

Azzolini, U. **Two cases of Tuerk-Stillings' syndrome, one of them of the inverted type with preserved abduction and missing adduction.** Riv. di oftal. **2**:12-21, Jan.-Feb., 1947.

The syndrome of Tuerk, called syndrome of Stilling by French authors, consists of unilateral absence of abduction, while adduction of the same eye is restricted and accompanied by enophthalmos and narrowing of the lid fissure. Discussing the numerous explanations proposed in the literature, Azzolini prefers the assumption of multiple anomalous muscle insertions for his cases. (12 photographs, bibliography.) K. W. Ascher.

Burian, H. M. **Sensorial retinal relationship in concomitant strabismus.** Arch. Ophth. **37**:504-533, April, 1947.

This is a continuation of a paper on the same subject. The clinical picture of anomalous retinal correspondence and its interpretation is discussed with the presentation of illustrative case reports. Not all patients with concomitant strabismus develop anomalous correspondence. The first prerequisite is a certain amount of binocular cooperation.

The principles of treatment of anomalous correspondence are outlined. Under this heading are discussed occlusion, surgery to restore normal position, prisms, and orthoptics. In discussing surgery, the author states that every child with strabismus should have his eyes straightened before entering school or shortly thereafter. (To be concluded.)

John C. Long.

Coachman, E. H. **A new syndrome, the association of latent vertical phoria with endocrine dysfunction.** South. M. J. **40**: 656-665, Aug., 1947.

A series of cases is presented in which the patients were made more comfortable after refraction when special stress was put upon vertical imbalances. These defects are brought out or made more evident by total occlusion of one or the other eye for 48 hours and measured with the usual Maddox rod.

The author has noted an association of symptoms, namely scanty eyebrows in the outer third, subnormal temperature, low blood pressure and slow pulse, such as are commonly found in decreased activity of the thyroid. There is a generalized edema especially of the lids and face, and along the medial aspect of the lower part of the tibia.

The patients commonly complain of periodic headaches which are usually unilateral and occur around or between the

eyes and radiate to the occiput, down the neck and into the scapular areas. The headaches are apt to follow movies, sewing, reading, card playing or driving, and may even be present upon awakening. Sensitivity to changes in position or motion is prominent. Children are poor readers, or skip lines entirely. Wearers of dark glasses are apt to have vertical imbalances, and when corrected by proper prisms are made comfortable with clear glasses. A. G. Wilde.

Neely, J. C. **The Treatment of traumatic diplopia.** Brit. J. Ophth. **31**:581-642, Oct., 1947.

This dissertation is in its essentials an apologia for active and more radical treatment of the diplopia which has been shown to be a not infrequent result of an injury to the head or face. The author discusses diagnosis and treatment and documents his opinion by liberal quotations from the records of 55 patients, members of the Royal Air Force personnel. The illustrations include reproductions of photographs, X-ray films and Hess charts. Diplopia is usually caused by a paresis and not a paralysis of an extraocular muscle and the vertically acting muscles are most often involved. In the majority of cases the "closed" head injury and not the penetrating wound is the cause of diplopia. Injuries to the orbit tend to be overlooked owing to the severity of other injuries, yet the restoration of the normal anatomy of the parts is of immediate importance. Well planned eye muscle surgery aided by orthoptic exercises is the best means of restoring binocular single vision. Once the vertical deviation has been reduced by operation to manageable proportions, the binocular reflexes strengthened by orthoptic training will enable the patient to maintain fusion within the range of normal ocular movement. If the deviation is considerable it is best to carry out the correction in stages rather than risk overcorrection and another relative paresis which may prove more troublesome to patient and surgeon than the first. O. H. Ellis.

Wright, E. S. **Bilateral ophthalmoplegia in acute anterior poliomyelitis.** Am. J. Ophth. **30**:1294-1298, Oct., 1947. (1 figure, 16 references.)

7

CONJUNCTIVA, CORNEA, SCLERA

Attanasio, V. **Epidemic keratoconjunctivitis.** Boll. d'ocul. **25**:481-500, Oct.-Dec., 1946.

Five cases of keratoconjunctivitis that occurred in patients whose ages ranged between 16 and 34 years are reported in detail. The disease was monocular and characterized by initial swelling and redness of the caruncle and the plica which was followed by the appearance of follicles on the fornix and palpebral conjunctiva. The pre-auricular glands were swollen. Photophobia and profuse tearing were among the common symptoms. Corneal lesions lasted a few months and left more or less defective vision. The usual course of the disease ranged from two to four weeks. The etiology and the treatment are discussed. The instillation of lemon juice combined with sulfamide therapy shortened the course. (4 figures, bibliography.) Melchiore Lombardo.

Bartolozzi, R., and Leoz de la Fuente, G. **Fatty degeneration of the cornea.** Arch. Soc. oft. hisp.-amer. **7**:558-564, June, 1947.

The authors describe three cases of this rare affection, of which only 15 cases have been reported. A woman 40 years old, had a primary corneal peripheral lipodystrophy. A man, 24 years of age, sustained a burn of the right cornea in infancy, and

this eye was much more extensively involved than the left. The third patient had congenital aniridia, coronary cataracts and a peripheral bilateral lipodystrophy. Visual acuity was severely impaired in all three patients. None of them had a demonstrable disturbance of the cholesterol metabolism. Their ingestion of fat was limited by economic conditions. The lipodystrophy was therefore attributed to a disturbance in the local oxidation which transformed invisible cholesterol into a visible form. The literature is reviewed, and it is noted that therapy is ineffective. (7 illustrations.) Ray K. Daily.

Castelli, A. **Keratoplastic surgery.** Rassegna ital. d'ottal. **16**:253-269, July-Aug., 1947.

The author performed 12 transplantations of corneal grafts, of which 11 healed with clear transplant. Six of the patients had keratoconus, 2 congenital familial degeneration of the cornea, and 4 had corneal complications of trachoma.

The technic was essentially that used in the United States. The author uses a 5-mm. trephine and advises the operation particularly for keratoconus and for corneal scars that are not total or vascularized. Eugene M. Blake.

Chace, R. R., and Locatacher-Kohorazo, D. **Keratoconjunctivitis due to a diphtheria-like organism.** Arch. Ophth. **37**: 497-503, April, 1947.

A woman, 26 years of age, had a bilateral keratoconjunctivitis for approximately seven years. The tissue of the mucocutaneous juncture of the lids was replaced by a scar and trichiasis was severe. There was roughening and redness of the conjunctiva of the fornices with papillary hypertrophy. The corneas showed a thin vascularized membrane with some staining with fluorescein. The patient was somewhat undernourished. Very detailed studies by culture and animal inoculation identified the causative organism as one of the Corynebacterium xerose group. Treatment has consisted in the removal of the cilia, sulfathiazole ointment and penicillin ointment. In spite of clinical improvement, the organism still appears in heavy growths in culture.

John C. Long.

Chavarria Iriarte, F. A. **Progressive, circular, bilateral fibro-vascular degeneration of the cornea.** Arch. Soc. oft. hisp.-amer. **7**:565-571, June, 1947.

The patient, a woman 61 years old, suffered from recurrent corneal marginal ulcers for a number of years. During this period of observation she developed a progressive bilateral circular pannus, which in some areas assumed the aspect of degenerative keratitis, with a slight corneal depression. A small zone of deep grayish infiltration in the cornea, without blood vessels, which extended beyond the vascularized area was biomicroscopically demonstrable. The differential diagnosis of corneal pannus is discussed. The etiology of the degenerative lesion in this case remains unknown. (3 illustrations.)

Ray K. Daily.

Chuliá, Vicente. **Five cases of Groenouw's corneal dystrophy in one family.** Arch. Soc. oft. hisp.-amer. **7**:572-577, June, 1947.

Groenouw's nodular corneal dystrophy occurred in the eyes of a man, 60 years old, and in the eyes of his mother, of her brother and of this brother's two sons. A slight increase in the blood calcium was found. No new data are presented. (3 illustrations.) Ray K. Daily.

Colombo, Giuseppe. **Four cases of keratitis superficialis vesiculosa disseminata, three of them associated with formation of corneal filaments.** Riv. di oftal. **2**:1-11, Jan.-Feb., 1947.

Keratitis filamentosa is not a disease entity; filaments may appear on the corneal surface whenever vesicles of the epithelium are formed as a result of trauma, inflammation or, degeneration. Vesicles of the corneal epithelium are broken and rolled by the lid movements to form the characteristic threads. Neurotrophic factors may be involved. (1 drawing.) K. W. Ascher.

Franklin, H. C. **Prophylaxis against ophthalmia neonatorum.** J.A.M.A. **134**: 1230-1235, Aug. 9, 1947.

Since Credé of Leipzig instituted the 2-percent silver nitrate prophylaxis of ophthalmia of the new born, that method has become mandatory in most countries although the concentration was reduced to one half because the original is uniformly irritating. Even this can cause a chemical conjunctivitis when undissolved crystals get into the eyes, or when the solution has become concentrated by evaporation.

Solutions of silver nitrate decompose slowly and the liberation of a small amount of nitric acid augments the irritating effect.

Ophthalmia neonatorum was once regarded as uniformly gonorrheal in origin but the term is now applied to any inflammatory process that appears within two weeks after birth. The staphylococcus, a diphtheroid bacillus, and pneumococcus may be the etiologic agent and the latter can be as destructive of the cornea as the gonococcus.

Believing that penicillin possesses the virtues but not the vices of the older agent, the author used it for a four month period. The crystalline sodium salt was employed in a concentration of 2,500 units per cc. in isotonic sodium chloride. The solution was not kept longer than one week, and when not in use was refrigerated below 59°F. Within one hour after delivery, secretions were removed from the lids, the conjunctival sac was washed with 2 to 3 cc. of isotonic sodium chloride, and one drop of the penicillin solution was instilled. This procedure was carried out in the nursery for three days.

It was found that the irritation of the eyes was much less after penicillin than after silver, and the prophylactic action of the former is just as effective.

A. G. Wilde.

Gallego Asorey. **Keratomalacia.** Arch. Soc. oft. hisp.-amer. **7**:469-476, May, 1947.

A typical case of keratomalacia is reported in a child two years old in whom nutritional deficiencies were found to be the cause. Twenty days after he was first seen the child died of a pulmonary disease. J. Wesley McKinney.

Gonçalves, Danilo. **Corneal ulcer in chickenpox.** Rev. brasil. de oftal. **6**:31-36, Sept., 1947.

The condition was seen 21 times in ten months. Such ulcers may have serious consequences as to vision. Prophylaxis by disinfection of the eyes and the nasopharynx is recommended. No case of associated depigmentation of the iris (false vitiligo) was seen. W. H. Crisp.

Kilgore, G. L. **The value of corneal peeling or corneal resection in ophthalmology.** Tr. Am. Ophth. Soc. **44**:100-112, 1946.

The author presents his method of corneal peeling or corneal resection. He first injects physiological solution along the line of initial incision to avoid danger of damage to the posterior corneal lamellae. Resection is of particular value where the scar is limited to the anterior half of the cornea. Visual results then compare favorably with those of keratoplasty. Aphakia is not a contraindication. Indica-

tions and contraindications are systematically listed. Keratoplasty can be performed if corneal resection fails. Several cases of successful corneal resection are described in detail. C. D. F. Jensen.

Laval, J., and Schneider, J. **Modified Snellen suture for prolapsed chemosed conjunctiva.** Arch. Ophth. **38**: 375-377, Sept., 1947.

The authors describe a case in which a new operative procedure, a modification of the Snellen suture for ectropion was used successfully in the treatmentof prolapsed chemosed conjunctiva of the lower eyelids. This procedure has the twofold purpose of restoring the prolapsed conjunctiva and of reforming the inferior fornix. (2 references.)

R. W. Danielson.

Leoz de la Fuente, G. **Superficial filamentous keratitis and lattice-shaped keratitis profunda.** Arch. Soc. oft. hisp-amer. **7**:553-557, June, 1947.

Leoz de la Fuente reports an unusual combination of an acute filamentous keratitis with a keratitis profunda in interlacing straiae as described by Haab and Dimmer, in an obese woman, 59 years old, with hypertension. That the cause was an infection is concluded from the prompt recovery in eleven days when 20-percent sulfathiazole solution was instilled after failure to improve with heat, dionin, and yellow oxide of mercury. The literature is reviewed. No new viewpoints are presented. (2 illustrations.)

Ray K. Daily.

McKinney, J. W. **Indication for corneal transplantation.** South. Surg. **13**:622-625, Sept., 1947.

Corneal transplantation is indicated where the superficial ulcers or burns have not affected the deep corneal stroma, deep ulcers or opacities involving the central portion of the cornea only, mild interstitial keratitis and keratoconus. It is not indicated in extensive ulcers, burns and deep infiltrating diseases, severe interstitial keratitis, trachoma with pannus, adherent leucoma, glaucoma, aphakia and the dystrophies. Irwin E. Gaynon.

McLaughlin, R. S. **Chemical burns of the human cornea.** Am. J. Surg. **74**:373-377, Sept., 1947.

The urgent immediate treatment of a chemical burn of the eye consists of immediate and copious irrigation of the eye with tap water. This should be done at the site of the accident. No neutralizing substances are used. The patient is then brought to the dispensary where the eye is anesthetized with 0.5-percent pontocaine solution, stained, particles removed, and the eye irrigated for fifteen minutes with normal saline. The eye is stained again with fluorescein. If it stains, the eye is again irrigated for 15 minutes. If the cornea still stains, the involved epithelium is removed by denudation using a round tooth pick swab, moistened with 4-percent cocaine hydrochloride.

Irwin E. Gaynon.

Newell, F. W. **Stimulation of corneal epithelization with topical application of erythrocytes.** Am. J. Ophth. **30**:1238-1243, Oct., 1947. (22 references.)

Petragnani, P. V. **Reiter's disease.** Riv. di Oftal. **2**:51-57. Jan.-Feb., 1947.

Petragnani describes the case of a man, 30 years of age, who had Reiter's disease twice in two years. Cultures taken from urethra, blood and conjunctiva did not reveal any causative organism. Recovery occurred in eight days after local antiseptic and stringent treatment supported by three intramuscular injections of sulfoidol. In another case, local treatment and sulfonamids and salycilates were of

no avail. Finally after three weeks recovery occurred after intramuscular administration of sulfoidol.

K. W. Ascher.

Reese, A. B. **Pigmentation of the palpebral conjunctiva resulting from mascara.** Am. J. Ophth. **30**:1352-1355, Nov., 1947. (5 figures.)

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Bitran, David. **Recovery from sympathetic ophthalmia.** Arch. chilenos de oftal. **3**:318-322, Nov.-Dec., 1946.

A healthy man of 32 years sustained a penetrating corneoscleral injury, with hernia of the iris and ciliary body. Prophylactic injection of 80,000 units of penicillin did not prevent the development of sympathetic ophthalmia in the second eye, which continued to get worse even after enucleation of the injured eye. In the course of 48 days, however, the sympathetic involvement completely recovered under further use of penicillin and intensive use of sulfadiazine. The sympathetic nature of the inflammation was confirmed by a histologic study of the enucleated eye. (3 figures.)

W. H. Crisp.

Esterman, Benjamin. **Choroideremia.** Arch. Ophth. **37**:716-721, June, 1947.

Choroideremia is always bilateral, occurs almost invariably in males, is usually associated with night blindness and sharply contracted visual fields, but often with relatively unimpaired central vision, and in the majority of instances occurs in persons with myopia of moderate degree. The lesion has often remained stationary.

A case of choroideremia was observed in a white man aged 29 years whose chief complaint was night blindness. Corrected visual acuity of the right eye was 20/200 and the left 20/30. Fields were reduced to a radius of five degrees in each eye. There was only a small island of choroid in each eye. The differential diagnosis between this condition and retinitis pigmentosa, gyrate atrophy, choroidal sclerosis and massive retinal gliosis is discussed. (1 colored plate.) John C. Long.

Poniedel, C. **Calcium deposits in the iris.** Brit. M. J. **2**:691-692, Nov. 1, 1947.

Calcium deposits in the iris were an interesting and perhaps important finding in a patient who became severely ill in October, 1944. Soon afterward his vision became blurred and examination revealed a chalky deposit in the anterior chamber and retina of the right eye. More than one year later fine deposits of calcium were found in the iris. The clinical studies, which included chemical examination of the patient's blood, made it seem probable that the primary disturbance was a chronic renal disease with secondary increase of calcium in the blood and not a primary hyperparathyroidism. It was interesting to note deposits in the iris and the observance of renal calculi.

F. H. Haessler.

Schaegel, T. F. Jr. **Bilateral granulomatous uveitis from the use of horse serum in rabbits.** Am. J. Ophth. **30**:1225-1237, Oct., 1947. (5 figures, 4 tables, 17 references.)

Sorsby, A., and Reed, H. **Local penicillin therapy of hypopyon formation with special reference to the use of subconjunctival injection.** Brit. J. Ophth. **31**:528-551, Sept., 1947.

The treatment of 66 cases of hypopyon associated with infected corneal ulcer, iritis, glaucoma and with herpetic or neuropathic corneal lesions is presented in detail. It consisted of repeated subconjunctival injections of 50,000 units of

penicillin usually dissolved in adrenalin and novocaine solution combined with instillations of penicillin ointments and drops and with general sulphonamide therapy. The use of subconjunctival penicillin was superior to all other treatment in eyes with bacterial infection. It had little effect on the herpetic or neuropathic lesions and in the hypopyon associated with simple iritis atropine instillation alone was sufficient.

The penicillin was injected every 6 hours for 12 injections. After that an ointment of penicillin, 100,000 units to the gram, was instilled every four hours day and night until recovery.

Morris Kaplan.

9

GLAUCOMA AND OCULAR TENSION

Barkan, Otto. **Cyclodialysis, multiple or single, with air injection—an operative technique for chronic glaucoma.** California Med. 67:78-83, Aug., 1947.

The author's operation is designed to create a larger dialysis with less trauma than is possible with the ordinary technique, and, secondly, to control hemorrhage. The resulting cleft is larger and permanent. The technique is described in detail. His injection of air into the chamber has been seen to stop an iris hemorrhage. An injection, if too great, can cause a rise of intraocular tension by blocking the outflow in the angle and by producing a seclusion of the pupil. Saline solution is made to replace some of the air. Extreme miosis is a preoperative essential. Maximal miosis should be maintained postoperatively to prevent closure of the cleft and the channel outlet. He further advises permanent daily use of a miotic to assure maintenance of the opening and channel. Francis M. Crage.

Bloomfield, S. **Lability test for chronic simple glaucoma.** Arch. Ophth. 38:368-374, Sept., 1947.

The need for a more reliable provocative test for simple glaucoma led the author to design his lability test which is based on the fact that the induction of a transient vascular congestion in the eye would tend to produce a significant elevation in tension that would be most pronounced if the normal controlling mechanisms were pathologically defective.

Basically, the lability test consists in the simultaneous application of the cold pressor test of Hines and Brown and the jugular compression test described by Schoenberg. First the tension of each eye is measured. The patient then places one open hand up to the wrist in ice water. Simultaneously, a blood pressure cuff previously placed loosely about the neck is inflated to a pressure of 50 to 60 mm. Hg. At the end of exactly one minute, the ocular tension is again recorded, with the hand still in ice water and the cervical pressure undiminished. Then the hand is withdrawn, the pressure cuff is removed, and the test is over unless a repetition is desired for purposes of accuracy. A study of its results when applied to a series of eyes with chronic simple glaucoma and to another group without that condition indicates that it is a highly reliable test. (3 figures, 3 references.)

R. W. Danielson.

Díaz Dominguez, Diego. **The etiopathogenesis of glaucoma,** Arch. Soc. oft. hisp.-amer. 7:477-507, May, 1947.

This article deals exclusively with primary chronic glaucoma. There is no experimental work which can elucidate its pathogenesis because glaucoma is fundamentally a human disease. The author identifies glaucoma with ocular hypertension and does not believe in the existence of glaucoma without hypertension. Cases of glaucoma without hypertension have been reported but in his opinion the

hypertension was not discovered because the patient's tension was not taken often enough to get a true measurement. He believes that glaucoma develops because an initial neurovegetative dystonia causes a circulatory disturbance. The result of the persistent circulatory disturbance is a venous stasis that leads to increased capillary permeability and production of exudates which block the drainage outlets. The author presents an extensive review of theories of the development of hypertension in glaucoma and especially the role played by the aqueous humor. (Bibliography.) J. Wesley McKinney.

Gát, L. **The effectiveness of a modified cyclodialysis.** Ophthalmologica **114**:106-118, Aug., 1947.

This is a report from the University Eye Clinic of Debrecen (Hungary) where, during the last 25 years, glaucoma surgery has consisted chiefly of iridectomies and cyclodialyses. Blaskovics' "inverted" technique has been modified as follows. The scleral incision is made anterior to the lateral instead of the superior rectus muscle. Most of the upper-temporal quadrant of the ciliary body is dialysed. During the dialysis the globe is fixated and the bleeding tendency reduced by placing a muscle clamp tightly on the tendon of the lateral rectus. The statistically presented results are difficult to evaluate because the absolute number of operations is small, the period of postoperative observation is short, and the criteria of failure or success are not clearly stated.

The author believes that the cyclodialysis is the most promising operation for glaucoma. Its advantages are the ease of performance, the applicability to all forms of glaucoma, the feasibility of repeated cyclodialyses on the same eye, the inherent freedom from serious operative complications.

Cyclodialysis also has disadvantages. The intraocular pressure remains high during the first two or three days after the operation. The acute and some of the chronic glaucomas, therefore, require a posterior sclerotomy combined with the cyclodialysis. Severe hemorrhages may occur (incidence not given). If the absorption is slow, the essayist aspirates the chamber contents on the fourth or fifth day. The central visual acuity may be temporarily impaired by the residues of the hemorrhage or by a refractive change in the direction of greater myopia. Retinal detachment has been reported but has not occurred in the author's series.

The author believes to have shown that cyclodialysis has lived up to the prediction of its originator Heine: "Optimal effects are achieved with the smallest and least dangerous operation."

Since completing this report the author has moved the site of the operation again. The scleral incision is now made in front of the inferior rectus and the inferotemporal quadrant is dialyzed. "The results have been encouraging."

Peter C. Kronfeld.

Grant, H. W. **The diagnosis of early glaucoma.** Am. J. Ophth. **30**:1276-1285, Oct., 1947. (9 figures, 1 table, 18 references.)

Gros, B. H. **Glaucoma in young persons.** Ann. d'ocul. **180**:366-375, June, 1947.

In all glaucoma three factors are important: the constitutional, the environmental, and the age-time factor. The author relates the progress of 17 cases of glaucoma, mostly in young persons. These illustrate the important effects of ocular and extraocular vascular permeability in the aggravation of glaucoma. In the aged this vascular degeneration is an arteriosclerotic process, but in the young

it is a neurovegetative disequilibrium involving especially the hypothalmus. Syphilis and other diseases as well as the excessive use of stimulants, alcohol, tobacco, and especially caffein may further accelerate this constitutional degenerative tendency. Records of prolonged uncontrolled destructive emotions and the shocks and frustrations of life, singly and in combination, run through the glaucomatous histories of these 17 persons of different ages. Chas. A. Bahn.

Holst, J. C. **A statistical study of glaucoma.** Am. J. Ophth. **30**:1267-1275, Oct., 1947. (2 tables.)

Kronfeld, P. C. **Contribution to the chemistry of the aqueous humor in the glaucomas.** Tr. Am. Ophth. Soc. **44**:134-142, 1946.

The author presents interesting statistical studies of the protein and ascorbic acid content of the aqueous humor of normal eyes and of eyes with glaucoma of the wide-angle type. He correlates these findings with the ascorbic acid content of the blood. There is a close parallelism between the ascorbic acid content of the blood and of the aqueous and no apparent relationship between the permeability of the blood-aqueous barrier and the ascorbic acid level in the aqueous. He concludes that in the normal eye as well as in the eye affected with deep-chamber glaucoma the ascorbic acid level of the aqueous is dependent upon the blood-ascorbic acid level. C. D. F. Jensen.

Magitot, A. **The intracranial origin of glaucomatous optic atrophy.** Ann. d'ocul. **180**:321-341, June, 1947.

Glaucoma and ocular hypertension are not synonymous. The field changes in primary glaucoma are reasonably explained only by postulating extraocular vascular lesions in the optic nerve and hypothalmus that are either spastic or otherwise obliterative. These lesions reduce the blood supply to specific sections of the retina until anemia temporarily or permanently prevents their function. The upper nasal quadrant is first involved in 60 percent and the lower nasal field in 20 percent. Glaucomatous field changes center around the disc and not the macula which is involved much later, if ever. The foundation of functional and structural changes that occur in glaucoma is to be found in the arrangement of the several sectors in the retina, disc, optic nerve and the chiasm as well as in their vascular supply. The vessels of the optic nerve, like those of the retina, branch dichotomously and are terminal vessels with practically no anastomoses. Hence there is no more collateral circulation in spastic or other obliterative lesions of the optic nerve than in the retina. The binasal hemianopic fields which are frequent in advanced glaucoma also occur in chiasmal lesions, especially in those of vascular origin. The mydriasis which usually accompanies glaucomas cannot be produced by fluid vitreous injections that increase the intraocular tension. The pupillary changes in glaucoma are essentially bilateral, even though but one eye is involved. Similar changes may be induced with electric shock or cardiazol treatment. These basically affect the vascularity of the hypothalamus. The hypothalamic center of Karpus and Kreidl which is apparently primarily involved affects pupillary dilatation and is very near the other autonomic centers, which are involved in lacrimation, accommodation, and indirectly in intraocular tension and glaucoma. This autonomic regulation involves not only the quantity and quality of nervous impulses but also vascularity and permeability.

It is difficult to dissociate hypothalamic and pituitary function. The relative fre-

quency of glaucoma in patients with definite evidences of pituitary disfunction has long been known. Only recently are the association of bodily neurovascular spastic and other obliterative lesions in integrated patterns being recognized. Irrespective of their bodily locations, angioneurotic edema in the skin and the chronic glaucoma of epidemic dropsy in India, have in common a dominant neurovascular factor in which apparently hypothalamic dysfunction is involved. The accepted field changes in primary glaucoma are evidence that the atrophy is descending, not ascending. This means that paracentral, peripheral and central loss of vision is not primarily intraocular, but due to vascular lesions in the optic nerve and hypothalamus. (8 figures, 61 references.) Chas. A. Bahn.

Marr, W. G. **The clinical use of diisopropyl fluorophosphate (D.F.P.) in chronic glaucoma.** Am. J. Ophth. **30**:1423-1426, Nov., 1947.

Sabata, J. **A case of inborn glaucoma observed during seven years.** Lekarske listy **2**:477-479, Oct. 15, 1947.

A girl of healthy parents had bilateral congenital glaucoma which was followed by great growth of the eyeballs, subluxation of the lenses and spherophakia. The child, now eight years of age, began to grow strikingly in height at the age of six years and now she displays other symptoms of Marfan's syndrome.

F. H. Haessler.

10

CRYSTALLINE LENS

Arjona, J. **Two thousand cataract operations.** Arch. soc. oft. hisp.-amer., **7**:429-444, May, 1947.

This is a brief analysis of 2,000 cataract extractions performed by the author. Tabulated data are presented. His observations are also given on the behavior of certain types of cataracts with particular characteristics such as: juvenile, morgagnian, myopic, diabetic and those complicated by iridocyclitis.

J. Wesley McKinney.

Atkinson, W. S. and von Sallman, Ludwig. **Mercury in the lens.** Tr. Am. Ophth. Soc. **44**:65-70, 1946.

A rose-brown, homogenous reflex was visible on the lens in a patient with chronic mercurialism. The clinical observations were supplemented with histological and histochemical studies on fixed specimens of the eyes removed at autopsy. Four control lenses were also studied histologically and histochemically. The microscopic appearance of elongated and amorphous deposits of black or dark brown pigment articles arranged with their long axes parallel to the surface of the lens is described. The control lenses did not contain such deposits. Spectrographic examination revealed that mercury existed among the trace elements in a value slightly less than five gammas. The chemical nature of the deposits was further confirmed with microchemical tests. Bellows suggests the distribution of the mercury in the lens may be explained, as in the case of copper, by the migration produced by the electric current within the eyeball. C. D. F. Jensen.

Barkan, Hans. **An evaluation of methods and results of cataract operations as performed in private and clinic practice at Stanford Hospital.** Tr. Am. Ophth. Soc. **44**:71-79, 1946.

The author presents a survey of 771 cataract operations performed at Stanford Hospital during the past five years. The extracapsular method was used in 584 or 76 percent of these operations and the intracapsular in 187 or 23 percent. A vision of 20/30 or better was obtained in 83

percent of the intracapsular operations and in only 69 percent of the extracapsular. Sixty-two (10 percent) of the latter required discission. Retinal detachment followed eight and infection five extracapsular extractions and did not occur after the intracapsular procedure. There was vitreous loss in 5 of the 187 intracapsular operations and in 6 of 584 extracapsular. Barkan emphasizes the need of statistical reports of completely unselected material. Cases with poor prognosis are segregated from the body of the statistics. C. D. F. Jensen.

Buschke, Wilhelm. **Acute reversible cataract in chicken due to various nitrocompounds.** Am. J. Ophth. **30**:1356-1368, Nov., 1947. (2 figures.)

Duehr, P. A., and Hogenson, C. D. **Treatment of subchoroidal hemorrhage by posterior sclerotomy.** Arch. Ophth. **38**: 365-367, Sept., 1947.

Diagnosis of the expulsive type of subchoroidal hemorrhage is not difficult. The sudden severe pain, pallor, clammy perspiration and nausea associated with a rapidly hardening eyeball are characteristic. This occurred in a patient while the sutures were being tied after cataract extraction. The authors performed a sclerotomy and the knife was thrust into the wound to release the blood. This was repeated at least six times. The eye healed with normal tension and visual acuity of 2/100. R. W. Danielson.

Falcão, Pedro. **Utilization of oral suction apparatus in cataract operation.** Arq. brasil. de oftal. **9**:131-138, 1946. Appears in English translation, Am. J. Ophth. **30**: 1428, Nov., 1947.

Falcão, Pedro. **Technique of cataract operation with the oral-suction cupping apparatus.** Am. J. Ophth. **30**:1428-1442, Nov., 1947. (2 figures.)

Hoskins, L. C. **Penicillin therapy of experimental infections of the lens and vitreous with Clostridium welchii.** Arch. Ophth. **38**:301-309, Sept., 1947.

In view of the failure of penicillin iontophoresis to check infection of the lens with Clostridium welchii, the present study was undertaken with direct injection of penicillin into the lens and the vitreous. Direct injection of penicillin into the lens and vitreous of rabbits controlled infections with Clostridium welchii when the therapy was instituted within three hours after the inoculation. Less favorable results were obtained when the interval between the infection and the treatment was increased to six hours. The beneficial effect in the three hour series was obtained with moderate damage to the structures of the eye. (15 references.) R. W. Danielson.

Kettesy, A. **Restoration of the chamber after intra-capsular extraction.** Brit. J. Ophth. **31**:572-575, Sept., 1947.

Delayed restoration of the anterior chamber is more frequent after intracapsular extraction than after extracapsular but it is less significant because it cannot be ascribed to particles of capsule or cortex left in the wound. The easily verified and remediable causes are: delayed healing, reopening of the wound, and choridal detachment.

Occasionally the anterior chamber does not form at all and this very serious sign seems not to be appreciated.

It can occur after a perfectly normal operation with a perfectly healed wound. On the twelfth or fourteenth day the cornea becomes hazy, the tension rapidly rises and the eye becomes blind. In 20 years in which he performed 250 extractions annually, Kettesy lost two eyes from this occurrence, and chance affected a cure in a third and gave a possible explanation. Both eyes of the patient were

operated on by uneventful intracapsular procedures. On the twelfth day the tension rose and despite medical treatment, both eyes seemed lost. Cyclodialysis was attempted and by accident the spatula injured the hyaloid membrane of the vitreous. The latter immediately streamed forth, pushed back the iris and filled the anterior chamber. The procedure was repeated on the second eye with the same result. Satisfactory healing was uneventful. The cause of the rise in tension is purely mechanical. The flat iris becomes plastered against the cornea and kept there by the inelastic hyaloid membrane. The aqueous is absorbed by the vitreous and this increases the pressure so that the iris is pressed more firmly against the cornea. Morris Kaplan.

Leoz, G. **Cataract extraction in high myopia.** Arch. Soc. oft. hisp.-amer. 7: 445-458, May, 1947.

When a cataract develops in a patient with a high degree of myopia the cataract is usually found to be one of the most complicated. Generally, there is damage in the retina, choroid and vitreous humor which makes the success of the operation very doubtful. The author recommends that whenever it is possible it is much better to perform this kind of cataract operation in two stages; a total iridectomy, followed by extracapsular extraction a month later.

J. Wesley McKinney.

Parry, T. G. W. **Post-operative security in cataract cases.** Brit. J. Ophth. **31**:569-570, Sept., 1947.

To obviate expected difficulties in post-operative care of an 80-year-old somewhat unreliable patient a complete conjunctival flap was tied with a purse string suture so as to cover the entire cornea after an ordinary intracapsular extraction. The eye was bandaged and the patient walked back to bed and remained ambulatory. The suture started to give on the third day and the whole cornea was clear on the fourteenth day. The procedure seemed so satisfactory that it was repeated on 11 more patients with very good results. All patients were ambulatory after 24 hours and all healed uneventfully.

Morris Kaplan.

deRoetth, A. **Influence of the suture on complications following cataract operation.** Arch. of Ophth. **38**:315-330, Sept., 1947.

Use of deep sutures in cataract operations is a great step forward in eliminating certain complications. The preplaced appositional corneo-scleral suture, combined with a conjunctival flap covering the wound, is considered the best method. A modification of the McLean suture is described which eliminates the difficulty of making the incision in the presence of a prepared conjunctival flap. One or two sutures can be used. A series of 150 unselected cases of cataract extraction with use of the Verhoeff suture method A, and a second series of 150 cases of extractions with use of the new suture are compared as to post-operative complications, degree of astigmatism and final visual results. It was found that the new suture was superior to the Kalt-Verhoeff suture in the rapid and solid closing of the wound, thus keeping low the incidence of shallow and flat chambers, wound rupture and postoperative prolapse of the iris and vitreous. (38 references.)

R. W. Danielson.

Samuels, Bernard: **Cataract complicating glaucoma.** Tr. Am. Ophth. Soc. **44**: 51-60, 1946.

The author presents the sixth and last part of a survey undertaken to record all microscopic changes that can take place in the lens as a result of lesions in other

structures of the globe. He describes 31 cases of cataract that were believed to have been caused primarily by glaucoma and includes interesting observations on the pathological changes that take place in the retina, iris, and ciliary body. He is unable to shed light on the high incidence of extensive retinal hemorrhage in no less than half of the cases of primary glaucoma. He leaves the question unsettled as to whether glaucoma had led to an impediment in the venous outflow or whether an impediment in the latter led to glaucoma. He points out that malignant glaucoma, particularly when the lens is tilted into the wound, is usually caused by a subchoroidal hemorrhage. In two cases in this series postoperative malignant glaucoma was evidently caused by nonexpulsive subchoroidal hemorrhage. Two other cases of malignant postoperative glaucoma without detachment of the choroid are believed to be definitely the result of a swollen and hardened vitreous body. An extensive cyclodialysis frequently favors the more rapid development of the cataract and may lead to an insidious iritis which is hard to counteract because atropine must be used cautiously. After trephining the ciliary processes are often fixed to the scar and extensive hemorrhage is apt to follow the section which may lead to membrane formation and possibly to sympathetic ophthalmia. (10 figures) C. D. F. Jensen.

Seidenari, R. **The vortex and the posterior epithelium in cataract.** Rassegna ital. d'ottal. **16**:293-311, July-Aug., 1947.

The author discusses the question of the growth of the anterior epithelium around the equator and into the posterior surface of the lens. He reports 10 cases, giving the clinical histories and a description of the microscopic changes. Especial attention is given to the general aspect and width of the posterior epithelium, the form, size and aspect of the nuclei, and the number of layers of cells. The epithelial cells passed over the equator in 82 percent of 50 cases. In the nine lenses in which this was not so five had mature or hypermature cataract and four immature. No definite conclusions can be drawn except that the overgrowth of epithelium is not characteristic of any type or age of cataract but it is most marked in complicated cataract. (4 figures.) Eugene M. Blake.

11

RETINA AND VITREOUS

Dollfus, M. A., Thiebaut, F., Daum, S., and Wolinetz, E. **Tuberous sclerosis of Bourneville.** Presse Méd. p. 380, June 4, 1947.

The authors report the case of a boy of 18 years with the characteristic tetrad of symptoms that consists of skin lesions, phakomatosis retinae, epileptic crises, and mental retardation. The skin lesions were nevi, vitiligo, and adenoma sebaceum. The epileptic attacks first appeared at the age of 15 years. The mental retardation had resulted in a mental age of 8 to 10 years and there was a simultaneous somatic retardation.

The patient had a visual acuity of 10/10 in each eye but fundus examination revealed prepapillary tumors and multiple pseudo-colobomatous atrophic areas. The lesions did not change during the three months that the patient was observed.

Phillips Thygeson.

Espildora Luque, C., and Schweitzer, A. **The ocular fundus in 78 autopsied cardiovascular patients.** Arch. chilenos de oftal. **3**:297-304, Nov.-Dec., 1946.

The ophthalmoscopic examination was negative in 18 percent of the cases. In cases where the age was less than 40 years, the ocular background was negative in 32 percent; of those above 40 years

the negative finding was in 12 percent. In both groups of patients the normality of the eyeground coincided with severe lesions of the cardiovascular-renal apparatus; the renal lesion predominating in the young, and aortic, myocardiac, cerebral, or occasional renal lesions in those of greater age. In young cardiovasculars the ophthalmoscopic lesions were generally more discrete. An exception to this was in malignant angio-nephrosclerosis, which was always accompanied by frank and serious changes of the ocular background. W. H. Crisp.

Fritz, M. H., Fennessy, T. M., and Cabrera, H. **Operative technique of vitreous replacement.** Am. J. Ophth. **30**:1221-1224, Oct., 1947. (2 figures, 5 references.)

Greear, J. N. **Interpretation of ophthalmoscopic findings in arterial hypertension.** Am. Practitioner **1**:640-642, Aug., 1947.

The author gives a brief historical discussion of the topic and recommends the classification of retinal lesions in hypertension advocated by the Americal Ophthalmological Society.

In pathological studies the author found that cottonwool patches were the result of localized areas of serous exudate in the deeper layers of the retina, or cytoid bodies in the nerve-fiber layer. The macular star figure was composed of collections of lipoidal histiocytes usually in the outer plexiform layer. The flame-shaped hemorrhages involved the nerve fiber layer, the more punctate and round hemorrhages the deeper layers. There is a short discussion of the ophthalmoscopic findings in the various types of hypertension. (References.) Bennett W. Muir.

Hallum, A. V. **Changes in retinal arterioles associated with the hypertensions of pregnancy.** Arch. Ophth. **37**:472-490, April, 1947.

This is a detailed study of the fundus changes in hypertensions of pregnancy including a review of the literature. At least 10 percent of pregnant women show some degree of late toxemia. It is stated that an examination of the eyegrounds by one experienced in their interpretation gives the most essential information concerning the management and course of the various toxemias of pregnancy. The obstetrician should do his own ophthalmoscopy, because he will often gain valuable information without extra expense and delay to the patient.

Acute hypertensive true toxemia of pregnancy is characterized by localized and generalized spastic constriction of the arterioles, the degree of which has been found to parallel closely the severity of the toxemia. The degree of spasm of the retinal arterioles indicates the degree of angiospasm in other parts of the body. The eyegrounds should be examined routinely during the early months of pregnancy to determine the status of the arterioles. Later in pregnancy, if hypertension develops, the frequent inspection of the arterioles may help determine the treatment to follow. John C. Long.

Koch, F. L. P., and Strong, P. S. **Lipemia retinalis: report of a case.** Tr. Am. Ophth. Soc. **44**:187-194, 1946.

The authors report a case of lipemia retinalis in a diabetic subject. It is the sixty-sixth case of lipemia retinalis to be reported three of which unquestionably occurred in patients without diabetes. Photographs of the eyegrounds and of the serum show the contrast between the conditions present at the peak of the episode and on the twenty-first hospital day.

C. D. F. Jensen.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
904 Carew Tower, Cincinnati 2

News items should reach the editor by the 12th of the month

DEATHS

Dr. Frederick G. Adolph Bardenheier, St. Louis, Missouri, died September 26, 1947, aged 65 years.

Dr. Edward T. Hornback, Hannibal, Missouri, died July 15, 1947, aged 83 years.

Dr. Bertil Thomelius Larson, Pontiac, Michigan, died July 20, 1947, aged 54 years.

Dr. Charles Porter Small, Chicago, Illinois, died September 25, 1947, aged 83 years.

ANNOUNCEMENTS

FLORIDA MIDWINTER SEMINAR

This year the University of Florida Midwinter Seminar in Otolaryngology and Ophthalmology will be held at the Flamingo Hotel in Miami Beach, from January 12th to 17th. The lectures in otolaryngology will be presented on the first three days and those in ophthalmology, the last three days. The registration fee is $25.

Among the outstanding ophthalmologists who will lecture are Dr. S. Judd Beach, Portland, Maine; Dr. William L. Benedict, Rochester, Minnesota; Dr. Daniel B. Kirby, New York; Dr. Peter C. Kronfeld, Chicago; and Dr. Dohrmann K. Pischel, San Francisco.

GEORGE WASHINGTON UNIVERSITY COURSE

From January 26th to 30th, The George Washington University School of Medicine, 1335 H Street, N.W., Washington 5, D.C., will conduct an intensive postgraduate course in ophthalmology.

Fundamental and practical instruction in ophthalmic problems will be presented. Among the topics to be considered are the following: anatomy of the eye, orbit, and optic pathways; congenital defects of the eye; examination of the eye; mydriatic and miotic agents; ocular findings in intracranial disease; the neuro-ophthalmologic examination; psychosomatic disorders; ocular findings in systemic diseases; preoperative and postoperative care for eye surgery; management of cataracts; diagnosis and surgical management of ocular deviations; tumors of the ocular bulb and adnexa; penetrating injuries of the eye; nonperforating eye injuries; localization and treatment of foreign bodies in the eye and orbit; lacrimal apparatus; uveitis; glaucoma; pediatric, geriatric, and tropical ophthalmology.

Among the distinguished ophthalmologists who will take part are: Dr. F. Bruce Fralick, University of Michigan; Dr. Murray F. McCaslin, University of Pittsburgh; Dr. Phillip Robb McDonald, University of Pennsylvania; Dr. Charles A. Perera, Columbia University; Dr. Raymond Pfeiffer, Columbia University; Dr. James S. Shipman, University of Pennsylvania; Dr. Isaac Samuel Tassman, University of Pennsylvania; Dr. Charles Dwight Townes, University of Louisville; Dr. Frank B. Walsh, Johns Hopkins University; Dr. Alan Woods, Johns Hopkins University.

Dr. Ernest A. W. Sheppard, professor of ophthalmology, is the faculty member in charge. The fee for the one-week course will be $50.

OREGON TO HAVE COURSE

The ninth annual spring postgraduate course in ophthalmology and otolaryngology will be held in Portland from March 21st to 26th. Another fine program has been arranged by the Oregon Academy and the University of Oregon Medical School. Dr. A. D. Ruedemann, Wayne University, Detroit, and Dr. C. Allen Dickey, University of California, San Francisco, will be among the guest speakers. There will be lectures, clinical demonstrations, and ward rounds.

Preliminary programs will be ready about February 1st. A program and further information may be obtained from Dr. Harold M. U'ren, secretary, 1735 North Wheeler Avenue, Portland 12, Oregon. In order to make the course more practical and personal, registration will be limited to 125.

A.O.S. TRANSACTIONS AVAILABLE

The American Ophthalmological Society offers back copies of its *Transactions* from its founding through 1939, with the exception of the following numbers that are not available: vol. 1, 1865, 1867, 1868; vol. 6, part 3, 1893; vol. 8, part 2, 1898; vol. 16, 1918; vol. 33, 1935; vol. 34, 1936; vol. 35, 1937. These volumes have been carefully stored and are in good condition.

Volumes 1 through 15 (years 1866 through 1917), vol. 21 (1923) and vol. 26 (1928) are unbound at $3 each. The other volumes are in the standard green binding at $5 each.

Send orders to: Dr. Maynard Wheeler, M.D., 30 West 59th Street, New York 19, New York.

MISCELLANEOUS

CHICAGO REFRESHER COURSE

The Chicago Ophthalmological Society held a one-week refresher course from December 8th to 13th. The subjects covered were office procedure, ocular motility, gonioscopy, glaucoma, ocular pathology, ophthalmoscopy, neuro-ophthalmology, perimetry, slitlamp biomicroscopy, refraction, and medical ophthalmology.

Staffs of the four Chicago medical schools and of Cook County Hospital served as instructors.

Eye hospital dedicated

The Thigpen-Cater Eye Hospital, commemorating Dr. Charles A. Thigpen and Dr. Job T. Cater, was dedicated on November 2, 1947, at Birmingham, Alabama. Dr. Alston Callahan, professor of ophthalmology, The Medical College of Alabama, has been named director of the Hospital.

Rivista di Oftalmologia

Florentine ophthalmology, more or less stagnant since the transfer of the "Bollettino d'Oculistica" to Rome and as a result of the recent war, is undergoing a reawakening. Prof. Biagio Alajmo, director of the Institute of Clinical Ophthalmology of the University of Florence, has founded a new monthly ophthalmologic journal, the "Rivista di Oftalmologia," which appeared in January, 1946, published by L. Salpietra, Florence, Italy.

The editor of the "Rivista" is ably assisted by a group of well-known Italian ophthalmologists—V. Accardi, A. Bencini, G. B. Bietti, F. Caramazza, D. Cattaneo, V. Cavara, A. Contino, Q. Di Marzio, E. Federici, R. Gallenga, L. Guglianetti, G. Lo Cascio, L. Maggiore, V. Rossi, A. Santonastaso, S. Sgrosso, A. Rubino, M. Simonelli, and U. Azzolini. The "Rivista" includes in its pages original papers, reviews, therapeutic notes, and abstracts from Italian and foreign journals.

Volume I, which the writer of this note (Charles A. Perera), has had the pleasure of reading, contains excellent papers by many prominent Italian oculists. If this volume is a portend of the future, it is safe to say that the "Rivista di Oftalmologia" will make an enviable reputation in Italian ophthalmologic literature. American ophthalmologists greet this newcomer with best wishes for its continued success.

Eye-Bank Fellowships

The Eye-Bank for Sight Restoration, Inc., has granted fellowships for research in ophthalmology to the medical schools of Harvard and Yale universities. Dr. Thomas Duane has been chosen to work at the Howe Laboratory, Harvard University Medical School, investigating the metabolism of the cornea. Dr. David Freeman, who will work at Yale University School of Medicine, will carry on experiments in tissue transplantation.

Riggs Optical Company moves

The Chicago city and general offices of the Riggs Optical Company are now located at 18 South Michigan Avenue, Chicago, where the company occupies the 4th and 5th floors.

Societies

N.S.P.B. annual meeting

The National Society for the Prevention of Blindness held its 33rd annual meeting on December 12th, in the Russell Sage Foundation Building, 130 East 22nd Street, New York.

The principal speaker was Dr. C. E. A. Winslow, editor of the *American Journal of Public Health.* Dr. Winslow's subject was "Prevention of Blindness in the Public Health Program."

Activities of the National Society for the Prevention of Blindness during the past year were reviewed by Dr. Franklin M. Foote, executive director. Mason H. Bigelow, president of the society, presided.

Reading meetings

At the 70th (Academy) meeting of the Reading (Pennsylvania) Eye, Ear, Nose, and Throat Society, the following members reported: Dr. Paul C. Craig, Dr. William J. Hertz, Dr. Isaac B. High, Dr. James E. Landis, Dr. John J. Penta, Dr. Samuel A. Phillips, Dr. Robert E. Shoemaker, and Dr. John M. Wotring.

The 71st meeting of the society was held jointly with the Berks County Medical Society. The speakers were Dr. Samuel A. Phillips of Allentown, Dr. Arthur A. Bobb of Reading, and Dr. William T. Hunt of Philadelphia.

Milwaukee program

On the scientific program of the November 25, 1947, meeting of the Milwaukee Oto-Ophthalmic Society were the following speakers: Dr. George Roncke, who discussed "Intraocular Tumors"; Dr. Wendell Jones, "Injection Therapy of Oesophageal Varices"; Dr. Charles Finn, "Case Simulating Acoustic Neuroma"; Dr. Howard B. Morter, "Otogenic Intracranial Infection"; Dr. Milton Loring, "Exophthalmos: A Case Presentation"; Dr. Claire M. Flanagan, "A Problem in Diagnosis of Laryngeal Carcinoma."

Personals

Gives Schoenberg Lecture

Dr. Peter C. Kronfeld, Chicago, delivered the first Mark J. Schoenberg Memorial Lecture on December 1, 1947, at the New York Academy of Medicine. The subject of the lecture was "The Canal of Schlemm."

Has new address

Prof. Ida Mann has resigned her appointment at Oxford University and has returned to London where her address is 87 Harley Street, London W. 1.

Receives Mackenzie medal

Mr. Eugene Wolff, F.R.C.S., London, was presented with the William Mackenzie Memorial Medal in Glasgow on October 24, 1947.

Speaks on corneal transplantation

Dr. A. E. Maumenee recently discussed "Corneal Transplantation" for the members of the department of ophthalmology, Medical College of Alabama, at the Thigpen-Cater Eye Hospital.

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